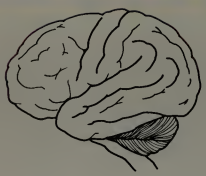
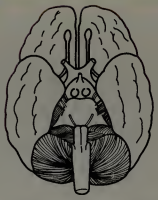
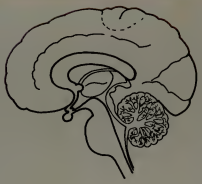
NEUROANATOMY

MADE EASY AND UNDERSTANDABLE

SECOND EDITION

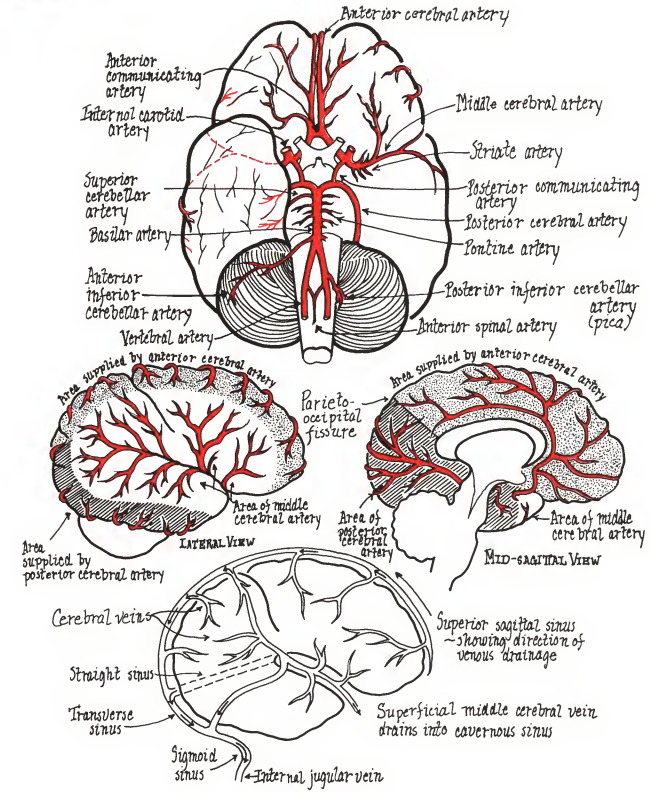
BY

MICHAEL LIEBMAN

UNIVERSITY PARK PRESS
BALTIMORE

Neuroanatomy Made Easy and Understandable

Second Edition


Anterior cerebral artery
Anterior communicating artery
Internal carotid artery
Middle cerebral artery
Striate artery
Superior cerebellar artery
Posterior communicating artery
Posterior cerebral artery
Basilar artery
Pontine artery
Anterior inferior cerebellar artery
Posterior inferior cerebellar artery (pica)
Vertebral artery
Anterior spinal artery
Area supplied by anterior cerebral artery
Parieto-occipital fissure
Area supplied by anterior cerebral artery
Area of middle cerebral artery
Area supplied by posterior cerebral artery
LATERAL VIEW
Area of posterior cerebral artery
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MID-SAGITAL VIEW
Cerebral veins
Superior sagittal sinus — showing direction of venous drainage
Straight sinus
Transverse sinus
Superficial middle cerebral vein drains into cavernous sinus
Sigmoid sinus
Internal jugular vein

Neuroanatomy Made Easy and Understandable

Second Edition

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Contents

Don't Skip This Introduction

Today you are faced with the problem of having to know more and more material in a shorter and shorter period of time. With neuroanatomy this problem is compounded because it is one of the most difficult subjects to grasp. Most "neuro" texts are very broad in scope and crammed with details, the latest theories, and so on. At this stage, however, you're unable to separate the wheat from the chaff, that is, to distinguish what is important for you from what is not. Consequently, you usually try to learn it all because you are afraid something will appear on the exam that you didn't read up on. Under conditions of high pressure and little time, this usually results in a monumental effort of memory, accompanied by little understanding and retention.

In this book I have cut out the fat of extraneous details, theories, and the like, and left *the essentials that form the basis for neuroanatomy, neurophysiology, neuropharmacology, physical diagnosis, and neurology, and for passing exams.* Although the subject is presented in a deceptively simple, breezy, and personal style, you must not assume that this was done by sacrificing material. The main reason for this approach was to make the subject easier to read, understand, and retain. Therefore, once you know the material in this book, you will be able to read and quickly understand more detailed neuroanatomy texts and reference books, should the need arise.

The terminology can throw you for two reasons. First, it is often redundant. For example, a group of nerve fibers may be called a tract, fasciculus, column, lemniscus, funiculus, or bundle—all terms accepted and used by the medical and scientific community. Second, the terminology is full of weird sounding names of Greek and Latin origins. As for the first, the author obviously cannot at his whim cut out recognized terms, but he can point out those that are synonymous. As for the second problem, I have prepared a special glossary that not only explains the meaning and origin of the names but also lists a common everyday word derived from them. For example, *fornix* is a Latin word meaning an arch and is applied to a curved bundle of nerve fibers. The related everyday word is fornication, and the reason for this is that in ancient Rome the prostitutes used to hang around the arches of the aqueducts!

I strongly recommend that you read each chapter before going to each lecture; then, instead of furiously trying to write down every word, you'll be able to sit back, absorb, and understand the material and leisurely jot down additional notes and drawings.

This second edition contains an entire new section covering pathologic conditions of the central nervous system. There is also a new appendix featuring CT scans of the brain showing normal anatomy and several common pathologic conditions. A number of additional diagnostic tests, clinical remarks, and historical notes have also been provided throughout the book.

Finally, I would welcome and appreciate suggestions and criticisms.

Good luck!

This book is dedicated
to my parents and
teachers

Chapter 1

THE MICROSCOPIC BASIS OF NEUROANATOMY

The basic unit of the nervous system, as in all other systems of the body, is the cell, which here is called the *neuron.* The main properties that distinguish neurons from other types of cells are their specialization for conduction of impulses, their great sensitivity to oxygen deprivation, their importance for many vital functions, and the fact that they don't multiply. (It is this last fact that is responsible for so many of the incurable conditions that you will see—paralysis, chronic vegetative states, palsy, blindness, etc.) This text discusses many types of neurons, and they all have the above-mentioned characteristics.

A typical neuron (Figure 1) consists of a cell body with a large *nucleus* that has a dark central *nucleolus.* Fine particles, known as *Nissl granules,* are scattered throughout most of the cytoplasm. Projecting from the cell body are many short processes—the *dendrites*—which receive impulses from other neurons and conduct them to the cell body. From the cell body a single long process—the *axon*—conducts the nerve impulse away and out to the dendrites of other neurons and to muscles and glands.

The site of contact between the axon of one neuron and the dendrites of another is the *synapse.* The impulse does not pass directly from neuron to neuron, however. Rather, it is transmitted by chemical mediators called *neurotransmitters.* The most widespread of these is *acetylcholine;* others include epinephrine, dopamine, and gamma-aminobutyric acid (GABA). The basic mechanism is as follows: the nervous impulse, which is measurable with fine instruments, travels down the axon until it reaches the synapse. Here it causes the release of neurotransmitter from the end of the axon, and this passes through the ultramicroscopic synaptic gap to the adjacent dendrites, where it triggers a new impulse that is then propagated in the second neuron (Figure 1). A single axon may synapse with the dendrites of several neurons, and the dendrites of one nerve cell can receive impulses from the axons of many neurons. Finally, there can be a combination of these two situations (Figure 2).

The axons of nearly all neurons are covered with a fatty white substance called *myelin;* in order for most impulses to be propagated, myelin must be present. In infants, myelin has not yet been laid down completely, and therefore they are unable to walk. In certain diseases, such as multiple sclerosis, the myelin degenerates and the patient suffers from a loss of various sensations and/or a diminution of movements. The process of *myelinization* (laying down of myelin) is performed by special cells that form an outer enveloping layer around the axon. This layer is known as the *sheath of Schwann (neurolemma).* Myelin is not a continuous layer but has gaps—the *nodes of Ranvier*—and here the overlying sheath of Schwann dips down and comes in contact with the axon (Figure 1).

Functionally and structurally there are many kinds of neurons; several of the most common are shown in Figure 3. A *motor* or *efferent neuron* is one that transmits impulses to muscles and/or glands, whereas a *sensory* or *afferent neuron* propagates sensory impulses. The nervous tissue of the brain and spinal cord is divided into *gray matter,* which is composed of the nerve cell bodies, and *white matter,* which is made up of the white axon fibers. Furthermore, nervous tissue has special cells—the *glia*—which are divided into three types: the first are *astrocytes,* whose functions are to hold together the delicate neurons and to help create the blood-brain barrier that prevents many substances from leaving the capillaries to enter the brain tissue; the second are the *microglia,* which act as scavengers *(phagocytes);* and the last are the *oligodendroglia.* Because the axons within

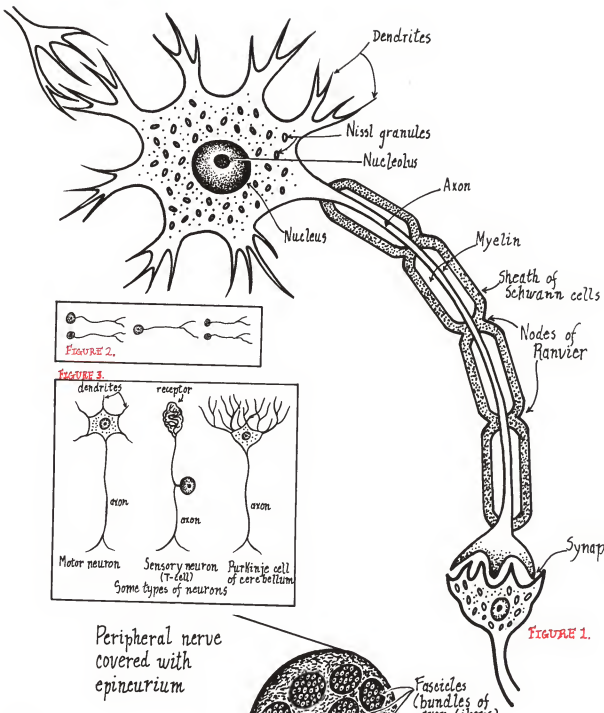


FIGURE 1.

FIGURE 2.

FIGURE 3.

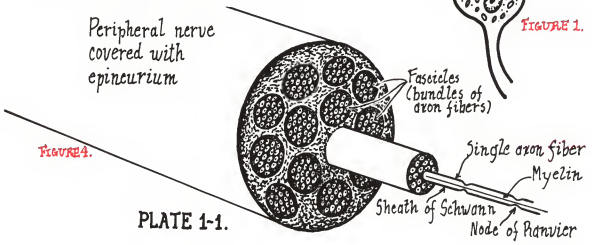


FIGURE 4.

PLATE 1-1.

the spinal cord and brain do not have a sheath of Schwann, it is thought that the oligodendroglia in these areas lay down the myelin. In addition to neurons and glia, there are the *ependymal cells*, which line the central canal of the spinal cord as well as the *ventricles*, and which are the first cells to appear during the embryonic development of the nervous system.

Although the nerve pathways in the figures in other chapters are represented by a single axon, this is artistic license for the sake of clarity. In reality every nerve and pathway is made up of many bundles, called *fascicles*, which in turn are made up of hundreds and hundreds of axons (Figure 4).

CLINICAL ASPECTS

Because mature neurons do not multiply, they are unable to give rise to brain tumors. The great majority of *neoplasms* (tumors) of the nervous system arise from glial cells or from the proliferation of other tissue cells found in conjunction with the brain, such as connective tissue or the epithelial cells of the pituitary gland. Very rarely, neurons still in an immature state will give rise to tumors known as *neuroblastomas*.

When a nerve is cut, a series of characteristic reactions takes place. That part of the axon distal to the injury quickly breaks down and dies, in a process known as *Wallerian degeneration*. The section of axon still attached to the cell body initially undergoes some degeneration, but if the damage isn't too extensive, it will start growing. However, its growth is inhibited by the rapid proliferation of Schwann cells, which form a dense scarlike mass. In cases where the severed ends of a nerve are sewn back together, growth will occur and there will be some renewal of normal function. The amount of renewal depends on such factors as the degree and location of injury, the quickness and skill of repair, the amount of glial cell proliferation at the repaired site, and the age of the patient. Unfortunately, axons within the brain and spinal cord cannot grow if they are severely damaged or cut.

Multiple sclerosis is a fairly common neurologic disease affecting mostly young adults in which the myelin breaks down, producing a variety of symptoms such as a reduction or loss of sensations, loss of motor ability, or double vision. Indeed the variety and mixture of symptoms is one of the most important clues in diagnosing the illness. The cause of the disease is unknown, and at the present time there is no known cure or means of prevention. Strangely, the myelin often forms again and the symptoms disappear until the myelin degenerates once again. A patient may have a few such episodes and then recover completely, or the attacks may progress and spread rapidly ending in death. In many other individuals the attacks and remissions last for many years.

One of the most puzzling things about multiple sclerosis is that in tropical areas the incidence of the illness is 1 per 100,000, whereas in colder areas such as Canada and Northern Europe the incidence soars to 30–80 per 100,000. The explanation for this fact may hold the key to the cause and cure of the disease.

Chapter 2

THE MACROSCOPIC BASIS OF NEUROANATOMY

This chapter deals with the macroscopic, many-named areas of the brain—a topic that can be very boring. However, it is necessary because one can't proceed with the study of the nervous pathways without knowing where they start, through what structures they pass, and where they end.

The nervous system is divided arbitrarily into a central and a peripheral part. The *central nervous system* (CNS) consists of the *brain* and the *spinal cord. The peripheral nervous system* is made up of the 12 pairs of cranial nerves and all the remaining nerves of the body and their associated collections of cell bodies—the *ganglia.*

FIVE PARTS OF THE BRAIN

The brain is divided on an embryologic basis into five parts: *telencephalon, diencephalon, mesencephalon, pons* and *cerebellum,* and the *medulla oblongata.* In this chapter we consider these five parts one by one.

Telencephalon

The telencephalon is the center for the highest functions and is therefore the most developed in humans. It is composed of two major structures: the *cerebral hemispheres* and the *basal ganglia.* The latter, which are the areas for crude motor activity, are buried deep in the cerebral hemispheres and can only be seen when the brain is cut. The cerebral hemispheres, on the other hand, are two very large structures divided from each other by the *median longitudinal fissure* and comprise most of the brain matter that is seen (Figure 1).* Their convex surface is made up of convolutions called *gyri,* which are separated from each other by shallow grooves—the *sulci* (a deep sulcus is called a fissure). Although certain gyri and sulci are present in almost every human brain, no two brains or even hemispheres of the same brain have exactly the same pattern of gyri and sulci. Two grooves, the lateral *fissure* and the *central sulcus,* help divide each hemisphere into four main areas, or *lobes* (Figure 2). The *frontal lobe* is anterior to the central sulcus, and the *parietal lobe* is posterior to it (Figure 2). Lying below the *lateral fissure* is the *temporal lobe,* and an imaginary line drawn down from the *parieto-occipital fissure* separates the parietal lobe from the *occipital lobe* (Figure 2). As if there weren't enough divisions already, each lobe has its specific areas and gyri. For example, in the frontal lobe the *precentral gyrus,* lying just anterior to the central sulcus, is the motor center that initiates impulses to the voluntary muscles. The most anterior area, the *frontal pole,* is the seat of personality (Figure 3). Injuries here often result in alterations of personality. These and other areas are discussed later in great detail.

The telencephalon also occupies much of the base of the brain. Here are situated the *orbital gyri,* and resting on them are found the *olfactory nerves,* which are the nerves of smell, and the *optic nerves,* which transmit visual impulses from the eye to the brain (Figure 4). The optic nerves converge on each other, cross at the *chiasma,* and then proceed posteriorly as the *optic tracts* (Figure 4). This view of the telencephalon also reveals the *parahippocampal gyrus* of the temporal pole with its characteristic bulge, the *uncus.*

When the brain is cut in a horizontal plane, one sees that the cerebral hemispheres have an outer gray layer, the *cortex,* which is composed of cell bodies, and an inner white mass made up of myelinated axons (Figure 5). Axons that pass from one hemisphere to the other are called *commissural fibers;* the best example of this is the large

*In addition to the drawings in this chapter and in Appendix II, the reader should refer to the CT scans in Figures 1–5 in Appendix III.

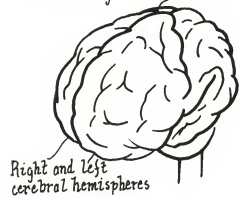
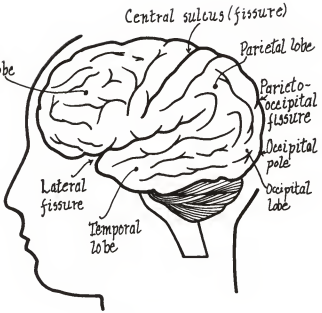


FIGURE 1.



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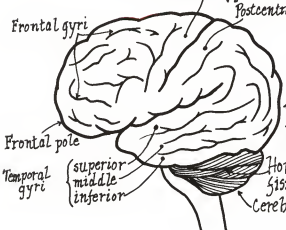

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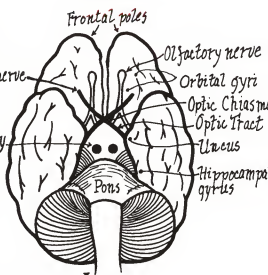


FIGURE 4.

PLATE 2-1.

corpus callosum (Figures 5 and 6). *Associative fibers* are those that pass from lobe to lobe or from gyrus to gyrus in the same hemisphere. Finally, those axons that descend from the cerebral hemisphere to other areas of the CNS are called *projection fibers,* and most are situated in the *internal capsule.* This structure has an *anterior limb,* a *posterior limb,* and a section between the two called the *genu* (Figure 5). Just lateral to the genu are located some of the *basal ganglia,* for example, the globus pallidus and the putamen (Figure 5).

Some neuroanatomy texts maintain that there are five lobes of the brain, whereas others claim there are six. If the lateral fissure is spread apart, one sees the *insula,* which is an infolding of the cerebral hemisphere. The insula has no known function in humans and is considered a fifth lobe. The cerebral cortex of the frontal, parietal, and temporal lobes surrounding the lateral fissure is known as the *operculum.* The limbic, or sixth lobe is made up of the *cingulate, parahippocampal,* and *dentate gyri.* (The author is waiting for the discovery of a seventh and maybe even an eighth lobe.)

Diencephalon

The diencephalon is the second division of the brain. It is a small area situated between the cerebral hemispheres and is seen best on a midsagittal view (Figure 6). The diencephalon is divided into the *thalamus,* which is the main relay center for the nervous system, and, below it, the *hypothalamus* (Figure 6). The hypothalamus is a vital area concerned with temperature control, emotional states, and control over the autonomic nervous system. In addition, the diencephalon is made up of the *medial* and *lateral geniculate bodies,* the *subthalamic nucleus,* and the *pineal body.* (Plates IV and IX in Appendix II, "Atlas of the Brain").

Mesencephalon

The mesencephalon, the pons, and the medulla oblongata together form a wedge-shaped structure, the *brainstem,* which extends down from the base of the brain to the *foramen magnum* of the skull (Figure 6). The mesencephalon, or *midbrain,* is the smallest of the five divisions of the brain and is located between the diencephalon and the pons (Figure 6). The area above the *aqueduct of Sylvius* (cerebral aqueduct) is the *tectum,* which is made up of four rounded projections—the *corpora quadrigemina.* The upper two projections form the *superior colliculi,* and the lower two the *inferior colliculi.* In the body or *tegmentum* of the midbrain pass various fiber tracts. Also situated there are the *red nucleus,* the *oculomotor nerve* and its nucleus, and the *trochlear nerve* and its nucleus. At the base of the midbrain there is a pair of huge fiber bundles, the *crus cerebri* (or *basis pedunculi*), which is a continuation of the descending projection fibers of the internal capsule (Figure 6a). Finally, situated between the tegmentum and the crus cerebri is the *substantia nigra.* The latter plus the crus and tegmentum make up the *cerebral peduncle.*

Pons and Cerebellum

The pons and cerebellum together make up the fourth division of the brain (Figure 6). The cerebellum is a many-folded structure located under the occipital lobe and is concerned with equilibrium, muscle tone, and the coordination of muscle activity. It is made up of a flocculonodular lobe, two large hemispheres, and a midline portion, the *vermis.* Passing between the cerebellum and the underlying brainstem are three pairs of fiber bundles: the *superior, middle,* and *inferior cerebellar peduncles* (see Chapter 11). They are also known as the *brachium conjunctivum,* the *brachium pontis,* and the *restiform body,* respectively. The pons is located between the midbrain and the medulla and is separated from the overlying cerebellum by a cavity—the *fourth ventricle* (Figure 6). Through the pons pass various ascending and descending fiber tracts. Also in the pons are the nuclei of the fifth cranial nerve *(trigeminal nerve),* the sixth cranial nerve *(abducens nerve),* and the seventh cranial nerve *(facial nerve).*

Medulla Oblongata

The medulla oblongata is the last division of the brain. It becomes continuous with the spinal cord at the foramen magnum (Figure 6). Like the pons and midbrain, it contains ascending and descending fiber tracts, as well as the nuclei of cranial nerves VIII through XII. The respiratory and cardiac centers are also situated in the medulla.

SPINAL CORD

The spinal cord is a long cylindrical structure beginning at the foramen magnum and descending to about the level of the second lumbar vertebra (L_2). The cord serves as the main pathway (highway) for the ascending and descending fibers tracts that connect the peripheral and spinal

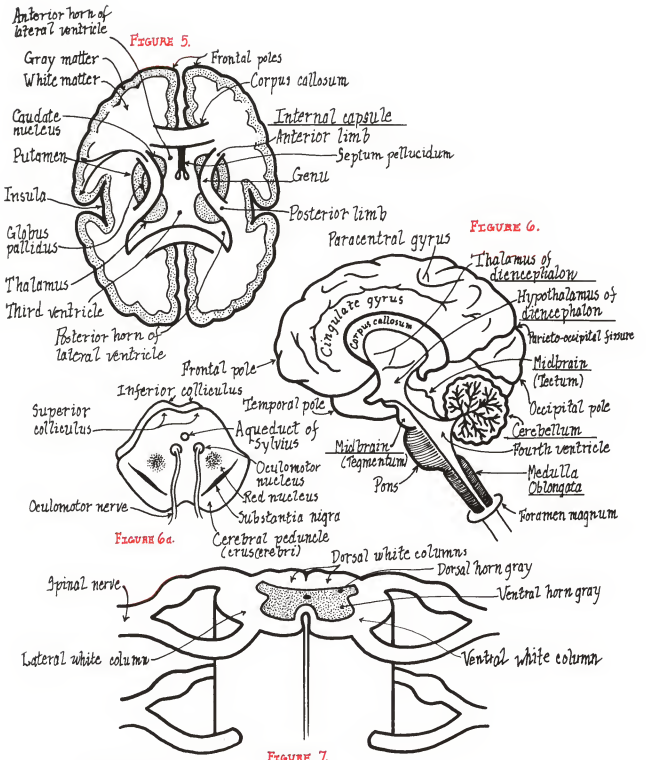


FIGURE 5.

FIGURE 6.

FIGURE 6a.

FIGURE 7.

PLATE 2-2.

nerves with the brain. The peripheral nerves are attached to the spinal cord by 31 pairs of spinal nerves.

A cross-section of the spinal cord reveals gray matter in the form of an H or butterfly surrounded on all sides by white matter. As in the cerebral hemispheres, the gray matter is composed of cell bodies, whereas the white is made up of the myelinated axon fibers (Figure 7). The upper limbs of the gray matter are the *dorsal* or *posterior horns*, and the lower are the *ventral* or *anterior horns*. The white matter is grouped into dorsal, ventral, and lateral *columns* (Figure 7).

After reading this chapter, some individuals may prefer to skip to Chapters 20–22 on the meninges, blood supply, and ventricular system before proceeding to the chapters on the various pathways.

Chapter 3

PAIN AND TEMPERATURE PATHWAY FROM THE EXTREMITIES AND TRUNK

Most people who consult a doctor do so because they are in pain. Therefore, a fundamental understanding of the pain and temperature pathway is essential for quick and accurate diagnosis. Fortunately, this is a simple pathway and a good grasp of it is easily obtained.

The receptors of pain and temperature are found in the dermis and epidermis of the skin. Nerve fibers pass from the dermis toward the spinal cord, with the cell bodies being situated in the dorsal root ganglion (Figure 1). The fibers then enter the cord through the dorsal root of the spinal nerve and end in the dorsal horn of the gray matter. Here the first neuron synapses with a second that then crosses to the contralateral (opposite) side of the cord, enters the lateral white column, and ascends to the ventral posterolateral nucleus of the thalamus (Figure 1; see also Appendix III, Figure 3). This ascending bundle of crossed pain and temperature fibers is known as the *lateral spinothalamic tract.* In the ventral posterolateral nucleus the axons of the lateral spinothalamic tract synapse with tertiary neurons that leave the thalamus and ascend in the internal capsule to reach the *postcentral gyrus* (Figure 1). The cortical gray matter of the postcentral gyrus (also known as area 3,1,2) is the primary somatic sensory area of the brain and is concerned with interpreting pain and temperature sensations, as well as other cutaneous sensations, such as pressure and touch (see following chapters).

ACCESSORY DETAILS

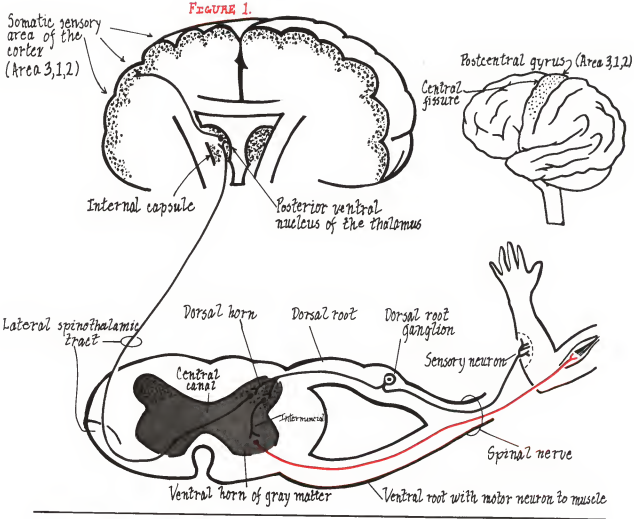
The primary pain and temperature axons have branches that synapse in the dorsal horn with short neurons that pass down to the ventral horn (Figure 1). Here these short *internuncial* (messenger) neurons synapse with motor neurons whose axons pass out through the ventral root and go out to voluntary muscles, causing movement. This involuntary motor response to a sensory stimulus is called a *reflex.* It is a defense mechanism of the nervous system that permits quick, automatic responses to painful and potentially damaging situations. The internuncials may cross over to the other side of the cord and stimulate motor neurons there, or they may descend or ascend the cord and stimulate motor neurons at different levels of the cord. It all depends on which group of muscles needs to be "called into action."

One interesting reflex involves the pupils of the eye, which dilate during severe pain. Thus, even though the patient may deny or not express his or her pain, the pupils will reveal it to the astute observer.

The dorsal root of the spinal nerve is composed of sensory (afferent) axons whose cell bodies are situated in the dorsal root ganglion. The ventral root, on the other hand, is made up exclusively of motor or efferent axons whose cell bodies are located in the gray matter of the ventral horn.

The fibers of each dorsal root come from a fairly circumscribed area of skin known as a *dermatome.* There is, however, at each boundary of the dermatome an area that is supplied by the adjacent segmental nerves. This overlap acts as a kind of biologic "insurance." For example, if the second thoracic nerve (T_2) is severed, then many of the pain and temperature sensations from the skin area supplied by T_2 will be carried by the T_1 and T_3 sensory neurons (Figure 2). There is also an overlap pattern in the spinal cord. The entering axon, before it passes into the dorsal horn, sends

FIGURE 1.



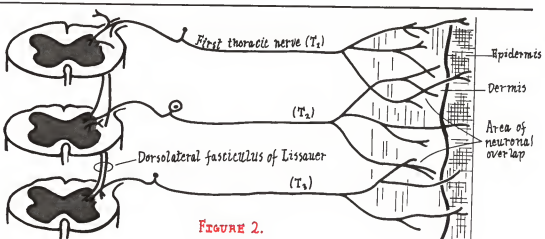


FIGURE 2.

PLATE 3-1.

branches that ascend and descend one spinal segment in the dorsolateral fasciculus (or column) of Lissauer and then enter the dorsal horn at that segment (Figure 2).

CLINICAL ASPECTS

The suffix -algia means pain; hence, neuralgia is pain in the nerves. An analgesic is something such as morphine, aspirin, or alcohol that deadens or dulls pain. (In the "Special Neuroanatomical and Neuropharmacologic Glossary" in Appendix I you will find interesting facts on various pharmacologic agents.) The suffix -esthesia, on the other hand, means sensations. Thus, an anesthetic is something that deadens or dulls all sensations.

A very common cause of neuralgia is pressure from an intervertebral disc upon a spinal nerve, which produces great pain. If this occurs in the lumbar area (lower back) the pain often radiates down the length of the leg and is called sciatica.

Referred Pain

The pain pathway from the viscera (internal organs) is poorly understood. Visceral pain is not well localized; in certain cases it isn't felt at the organ site, but is experienced at the surface of the body some distance from the affected organ. Such a reaction is known as referred pain, and in many instances it is quite specific and can serve as an excellent diagnostic aid. For example, a person suffering from a coronary attack often experiences a sharp pain that radiates along the inner aspect of the left arm; pain originating from the ureters is felt in the testes; pain from the lungs and diaphragm is experienced at the shoulders near the root of the neck. [An excellent discussion of the theories of referred pain is presented in Crosby's *Correlative Anatomy of the Nervous System*, page 83 (Macmillan, 1962).]

Phantom Limb

In many cases following amputation the patient complains of excruciating pain from the fingers or toes that no longer exist! The explanation for this strange phenomenon is as follows: A stimulus applied anywhere along the nerve fiber is experienced by the sensory cortex as coming not from the site of stimulation but rather from the skin area supplied by the nerves being stimulated. The nerve fibers at the stump are frequently squeezed by the scar tissue, and this pain stimulus passes to the sensory cortex, which interprets it as coming not from the stump area but from the skin areas of the fingers or toes of the missing limb.

Cordotomy

In cases of severe pain, as for example, from cancers or phantom limb, in which drugs no longer alleviate the pain, a surgical procedure known as *cordotomy* may be performed. The surgeon cuts the lateral spinothalamic tract of the cord, on the opposite side from the site of the pain and at a level one or two segments higher than the entrance of the uppermost spinal nerve that serves the affected area. The latter is done because of the overlap that exists in the cord (see above). In some cases the cordotomy is done on both sides.

DIAGNOSTIC TESTS

Pain

Have the patient close his or her eyes. With a pin, lightly prick the areas where pain sensations are thought to be absent. Ask when the patient feels the pin and when not.

Temperature

Take two test tubes, one with warm water and the other with cold. Again ask the patient to close the eyes. Alternately touch the test tubes to the areas where the sensations are believed to be lost and inquire whether the patient can feel the hot or cold.

Chapter 4

PATHWAY FOR PRESSURE AND SIMPLE (CRUDE) TOUCH FROM THE EXTREMITIES AND TRUNK

The receptors for pressure and crude touch are situated in the dermis of the skin. The nerve fibers travel in the peripheral nerves toward the spinal cord. The cell bodies are aggregated in the dorsal root ganglion, and from here axons enter the cord through the dorsal root (Figure 1). Upon entering, the axons pass into the ipsilateral (i.e., the same side) dorsal white column and bifurcate. One branch immediately enters the dorsal horn gray matter and synapses with a second (or secondary) neuron. The other branch ascends in the ipsilateral dorsal column for as many as 10 spinal segments and then enters the dorsal horn gray matter to synapse with a second neuron (Figure 1). In both cases, the secondary neurons decussate (i.e., cross over to the other side) and enter the ventral white column, where they form the ventral spinothalamic tract. This tract ascends to the ventral posterolateral nucleus of the thalamus, where it synapses with third (or tertiary) neurons (Figure 1), which then relay the pressure and crude touch sensations to the postcentral gyrus of the cortex, which is concerned with interpreting sensations.

CLINICAL ASPECTS

Because one branch of the first neuron synapses immediately with a second neuron, whereas the second branch ascends ipsilaterally for many segments, injuries to the spinal cord rarely result in complete loss of pressure and crude touch sensations. For example, if there is any injury to the spinal cord at point A in Figure 1 and the ventral spinothalamic tract is cut, one sees that the long ascending branch of the primary neuron bypasses the injury (on the uninjured side), and thus the sensations can still reach the postcentral gyrus. Naturally, if the sensory cortex, the internal capsule, or the thalamus is injured, then the pressure and crude touch sensations are lost on the contralateral side of the body.

DIAGNOSTIC TEST FOR SIMPLE TOUCH

Have the patient close his or her eyes. Then gently stroke the skin area with a wisp of cotton and ask whether the patient feels it or not.

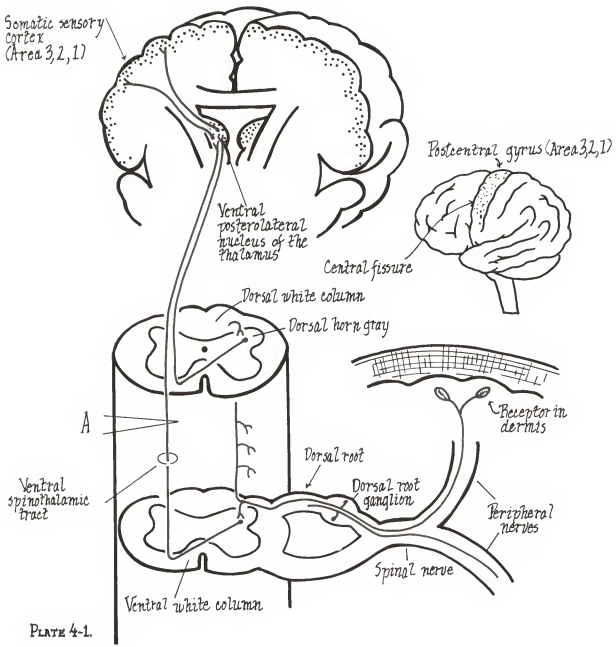

Somatic sensory cortex (Area 3,2,1)
Ventral posterolateral nucleus of the thalamus
Postcentral gyrus (Area 3,2,1)
Central fissure
Dorsal white column
Dorsal horn gray
A
Receptor in dermis
Dorsal root
Dorsal root ganglion
Peripheral nerves
Ventral spinothalamic tract
Spinal nerve
Ventral white column

PLATE 4-1.

Chapter 5

PATHWAY FOR PROPRIOCEPTION, FINE (DISCRIMINATORY) TOUCH, AND VIBRATORY SENSE FROM THE EXTREMITIES AND TRUNK

Three different sensations—proprioception, fine touch, and vibratory senses—all use the same pathway. *Proprioception* is the sense that enables one to know exactly and at all times where the parts of the body are in space and in relation to each other. Thus, it enables a person, with the eyes closed, to bring up the hand and touch the tip of the nose with the index finger. Its receptors are located in muscles, tendons, and joints. *Fine touch* is the sense that enables a person, again with the eyes closed, to identify various objects, such as keys, velvet, coins, and ping-pong balls, by touch. This property is known in medicine as *stereognosis.* Fine touch also involves the facility to discriminate between two points when being touched by both points simultaneously, as with the two points of a compass. These receptors are situated in the dermis of the skin, and they are most sensitive in the fingertips and lips and least sensitive on the back. *Vibratory sense* is, as its name implies, the sensation of vibrating objects.

The fibers of all three sensations pass toward the spinal cord in the peripheral nerves, and the cell bodies are aggregated in the dorsal root ganglion. From here, axons enter the spinal cord and immediately pass into the ipsilateral dorsal white columns, where they ascend all the way up to the medulla (Figure 1). Axons that enter the cord at the sacral and lumbar levels are situated in the medial part of the dorsal column, which is called the *fasciculus gracilis,* whereas those axons that enter at the thoracic and cervical levels form the more lateral *fasciculus cuneatus* (Figure 1). The axons of each fasciculus terminate in their respective nucleus in the medulla. The second-order neurons leave the nucleus gracilis and the nucleus cuneatus and cross over to the other side of the medulla, where they form a bundle known as the *medial lemniscus,* which ascends to the ventral posterolateral nucleus of the thalamus (Figure 1). Here the second-order neurons synapse with third-order neurons that pass up through the internal capsule to reach the postcentral gyrus (area 3,1,2), which is the primary cerebral somesthetic (somatic sensory) region (Figure 2).

CLINICAL ASPECTS

Damage to the postcentral gyrus, to the medial lemniscus, to the dorsal column, or to the cell bodies in the dorsal root ganglion results in several distinct clinical symptoms:

1. Astereognosis, or loss of ability to distinguish between objects through touch and manipulation.
2. Loss of the vibratory sense.
3. Loss of two-point tactile discrimination—when touched simultaneously with two points of a compass, the patient reports feeling only one.
4. A loss of proprioception, so that there is an inability to know where the limbs are. Therefore, such a patient looks down at the feet when walking, and at night would stagger or fall. When asked to stand erect with both feet together and eyes closed, the patient's body sways—a positive Romberg sign.

If the injury is bilateral, then, of course, the symptoms will be on both sides of the body. If, however, the lesion is on one side, then the symptoms will appear on one side only, depending on

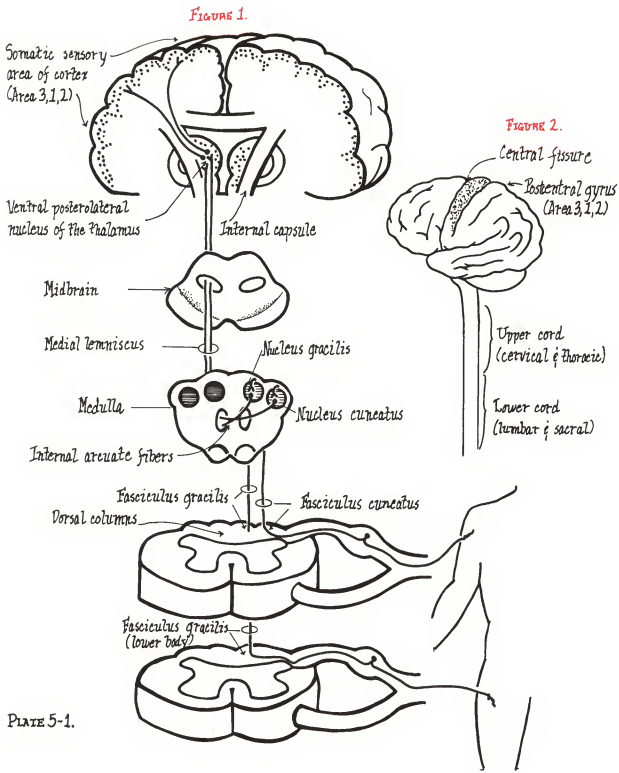


Figure 1.
Somatic sensory area of cortex (Area 3,1,2)
Ventral posterolateral nucleus of the Thalamus
Internal capsule
Midbrain
Medial lemniscus
Nucleus gracilis
Medulla
Nucleus cuneatus
Internal arcuate fibers
Fasciculus gracilis
Fasciculus cuneatus
Dorsal columns
Fasciculus gracilis (lower body)
Figure 2.
Central fissure
Postcentral gyrus (Area 3,1,2)
Upper cord (cervical & thoracic)
Lower cord (lumbar & sacral)

Plate 5-1.

where the damage is. If the damage is before the decussation—that is, in the dorsal root ganglion, the posterior column, or medullary nuclei—then the signs will be on the same side; if it is after the decussation—in the medial lemniscus, the thalamus, or cerebral cortex—then the signs will be on the side opposite to the lesion.

Damage to the dorsal root ganglion frequently occurs in the third stage of syphilis, when the bacterial organisms selectively attack and destroy the proprioceptive cell bodies but initially spare those of pain, temperature, crude touch, and pressure. Tabetics (those who have the third stage of the disease) therefore exhibit *ataxia*, a characteristic staggering and lack of coordination.

DIAGNOSTIC TESTS

Have the patient close his or her eyes. Then place in succession different objects (e.g. a key, a coin, a matchbox) in the patient's hand and have the patient describe the shape, size, and consistency of the object and to identify it.

With the patient's eyes still closed, ask him or her to touch the tip of the nose with the index finger, or have the patient stand erect and observe whether the patient sways when the eyes are closed. Failure to perform either task indicates proprioceptive impairment.

HISTORICAL NOTE

Syphilis has been present in the New World for thousands of years but it only appeared in Europe in 1495 at the siege of Naples. From there it spread rapidly throughout the Continent and became one of the most feared and prevalent diseases. The French called it the Italian disease, the Italians in turn called it the Spanish disease, the Spaniards called it the English disease, and so on. Therapy then consisted of spreading a cream containing mercury over the affected area for a period of several months. This gave rise to the joke that "you spend one night with Venus and six months with Mercury."* This scourge was finally cured with the discovery of Salvarsan by Paul Erlich. Salvarsan was also known as "606" because 606 experiments were done until he discovered it. Today penicillin is the drug of choice in the treatment of this illness.

*Mercury in those days was called "quacksalver," which later became "quicksilver," a word that is still used today. From "quacksalver" came the word "quack" or one who prescribes quacksalver (the treatment really didn't help very much).

Chapter 6

SENSORY PATHWAYS FROM THE FACE AND RELATED AREAS

Our discussions of somatic sensory pathways do not include nerves from the face and related areas because these areas do not "use" the spinal nerves. Sensations for these areas pass in the fifth cranial nerve, the trigeminal. The basic groundplan is pretty much the same, and an understanding of it is important, especially for those studying dentistry or who have chosen a specialty involving the cranial region.

The trigeminal nerve is the major somatic sensory nerve for the face, the anterior half of the scalp, the mouth cavity, the meninges, the sinuses, the teeth, the tongue, the cornea, and the outer surface of the eardrum. It transmits the sensations of pain and temperature and all kinds of touch, pressure, and proprioception, but not those of the special senses, such as hearing, taste, smell, vision, and equilibrium, which are carried by other cranial nerves.

PAIN AND TEMPERATURE PATHWAY

The pain and temperature pathway for the face and its adjacent regions is shown by the solid lines in Figure 1. From receptors situated in the abovementioned areas, fibers pass in the peripheral branches of the trigeminal nerve toward the brain. Their cell bodies are located in the *semilunar* or *Gasserian ganglion* (Figure 1), which is the analogue of the dorsal root ganglion. From here the axons enter the pons and are immediately concentrated in a bundle, the descending or spinal tract of cranial nerve V, which swings down and in many cases reaches the upper cervical region of the cord. Along this course the primary neurons peel off and enter the adjacent nucleus of the descending tract of cranial nerve V, where they synapse with secondary neurons. These leave the nucleus, cross over to the contralateral side, and ascend to terminate in the ventral posteromedial nucleus of the thalamus (Figure 1). This crossed pain and temperature bundle is called the ventral secondary ascending tract of cranial nerve V (or the ventral trigeminal tract) and is analogous to the lateral spinothalamic tract. From the thalamus, tertiary neurons pass into the internal capsule, ascend in it, and end in the postcentral gyrus (area 3,1,2)—the primary or main somesthetic region of the cortex.

PRESSURE AND TOUCH PATHWAY

These neurons (represented by the dashed lines in Figure 1) also have their cell bodies in the semilunar ganglion, but their axons terminate immediately in the main sensory nucleus of cranial nerve V, situated in the pons (Figure 1). The secondary neurons reach the ventral posteromedial nucleus of the thalamus via the dorsal secondary ascending tract of cranial nerve V (or the dorsal trigeminal tract) which is a crossed and uncrossed tract. That is, some axons travel ipsilaterally and some contralaterally and thus resemble the pressure and touch pathway for the body. Tertiary neurons are relayed from the thalamus to the postcentral gyrus (Figure 1). Thus we see that, whereas pain and temperature are projected on the contralateral cerebral cortex, pressure and touch are bilaterally projected. Therefore, if one side of the sensory cortex is damaged, the patient will suffer no loss of pressure or touch from the face, but will lose the pain and temperature feelings on the contralateral side.

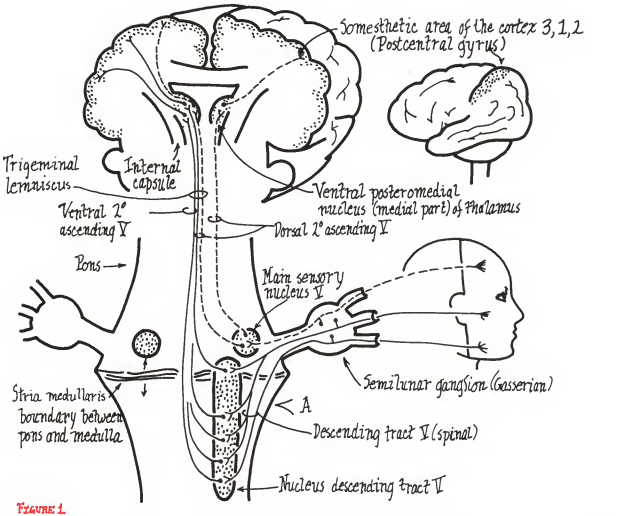


FIGURE 1.

FIGURE 2.

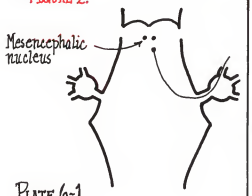


FIGURE 3.

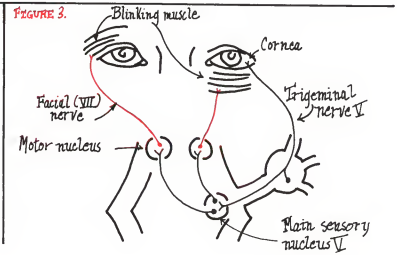


PLATE 6-1.

PROPRIOCEPTION PATHWAY

This pathway is composed of trigemino-proprioceptive fibers from the muscles of mastication and from the temporomandibular joint. However, they are an exception in that their primary cell bodies aren't in a ganglion outside the CNS but are situated in the mesencephalic nucleus in the midbrain (Figure 2). The further pathway of this sensation to the postcentral gyrus is not well known.

ACCESSORY DETAIL

There are several reflexes involving the trigeminal nerve, of which the most important is the corneal or "blink" reflex. If an object touches the cornea of one eye, both eyes will blink immediately. The pathway is as follows: the touch stimulus from the cornea reaches the ipsilateral main sensory nucleus of cranial nerve V. This sends out internuncial neurons that pass to the *right and left* motor nuclei of the facial nerve. From here motor neurons pass out and stimulate the muscles that cause blinking (Figure 3).

General sensations from small areas of the back of the ear and external auditory meatus are supplied by components of cranial nerves VII (facial), IX (glossopharyngeal), and X (vagus). The axons of these nerves enter the descending (spinal) tract of cranial nerve V and then follow the same pathway as the trigeminal fibers.

CLINICAL ASPECTS

Reflexes not only are a defense mechanism but also are useful diagnostically, enabling the physician to test the integrity of nerve pathways. If, upon testing, a reflex is not elicited, the physician then has to find out where the interruption in the pathway is: in the sensory pathway, the internuncial connections, or the motor pathway. In addition, there are reflexes that appear only in pathologic conditions, informing the physician that something is wrong.

When a person is anesthetized in surgery, specific reflexes disappear as deeper and deeper levels of unconsciousness are reached. Thus, the anesthetist is able to gauge accurately the level of unconsciousness by means of the presence or absence of these reflexes.

If the trigeminal nerve or the semilunar ganglion is damaged, then the individual will suffer loss of all facial sensations on the same side as the injury. As mentioned previously, injury to one side of the sensory cortex, internal capsule, etc., results in loss of facial pain and temperature sensation contralaterally, but pressure and touch will remain.

Trigeminal neuralgia (tic douloureux) is a condition of unknown etiology in which the patient suffers excruciating, shooting pain on one side of the face. Because drugs don't often bring relief, surgical treatment is used. The descending tract of cranial nerve V lies superficially, thus enabling neurosurgeons to go in and cut it on the side that has the pain (point A in Figure 1). Thus they sever the pain and temperature axons while sparing the pressure and touch fibers.

Brain tissue itself has no sensations whatsoever, and therefore operations on it can be done using only local anesthetics. Headaches are usually the result of pressure or pain in nonnervous structures on or within the brain or skull, such as the arteries or the meninges (the coverings of the brain).

Chapter 7

PATHWAY FOR VOLUNTARY MUSCLE ACTIVITY

Everyone has undoubtedly seen at one time or another individuals who can't walk and are confined to wheelchairs, or who walk slowly, dragging one leg, or whose arm lies helplessly flexed on one side; in short, persons who have some form of paralysis. In most of these conditions the muscles are fundamentally intact, and the condition is due to some kind of injury to the nerves. Because damage to no other pathway is responsible for so much suffering and sorrow, a first-class understanding of this pathway is mandatory.

The *corticospinal tract* is the main tract for nearly all voluntary muscle activity. It originates in the precentral gyrus (area 4, the motor cortex) of the frontal lobe. Here are located its large cell bodies. Since many of these have a pyramidal shape, the corticospinal tract is also called the *pyramidal tract.* (How a conscious wish is "translated" into cortical nerve impulses is an age-old question involving the mind-matter problem, and probably will never be answered satisfactorily.) From the cell bodies, axons leave the cortex and pass down through the internal capsule, which isn't a capsule but the main passageway for ascending and descending fiber tracts (Figure 1). Leaving the internal capsule, the axon fibers pass down into the basis pedunculi of the midbrain and continue down the brainstem to reach the medulla oblongata. Here about 80–90% of the axons decussate to the opposite or contralateral side of the medulla and, having crossed over, descend in the spinal cord (Figure 1). Because these descending fibers are situated in the lateral white columns of the cord they are called the *lateral corticospinal tract.* Those axons that do not cross over in the medulla continue down on the same side to enter the ventral white columns of the spinal cord and are therefore known as the *ventral corticospinal tract.*

At each level of the cord, axons from the lateral corticospinal tract peel off and enter the gray matter of the ventral horn, where they terminate by synapsing with second-order neurons. At each corresponding level of the cord, axons of the ventral corticospinal tract peel off and cross over to the other side of the cord (Figure 1). Here they also enter and terminate upon second-order neurons in the ventral horn. It must be emphasized that, in their entire course from the precentral gyrus to the ventral horn, both the lateral and ventral corticospinal tracts consist of single uninterrupted neurons; that is, the tracts are a single neuron pathway. These neurons are called *upper motor neurons.* The second-order neurons on which the upper motor neurons synapse send their axons out of the spinal cord via the ventral roots. They then branch out in the peripheral nerves and supply the voluntary muscles. These second-order neurons are *lower motor neurons,* and this differentiation between them and the upper motor neurons is very important clinically, as we shall soon see. In a person who is 6 feet tall, the axons that supply the toe muscles are nearly a yard long. The upper motor neurons begin in the precentral gyrus and end in the lower part of the cord, whereas the lower motor neuron begins in the lower cord and its axon passes down to supply the muscle situated on the sole of the foot.

DETAILS

Cerebral Localization

The nerve cell bodies of the upper motor neurons are arranged in a specific pattern in the gray matter of the precentral gyrus, so that neurons supplying the foot and leg muscles are situated dorsomedially in the gyrus. As one passes inferolaterally one finds the areas for the abdomen, chest, arm, hand, and face. On can describe this more colorfully by saying that the pattern is that of a person hanging upside down, with the feet in the longitudinal fissure and the head at the edge of the lateral fissure (Figure 1). The area of neurons

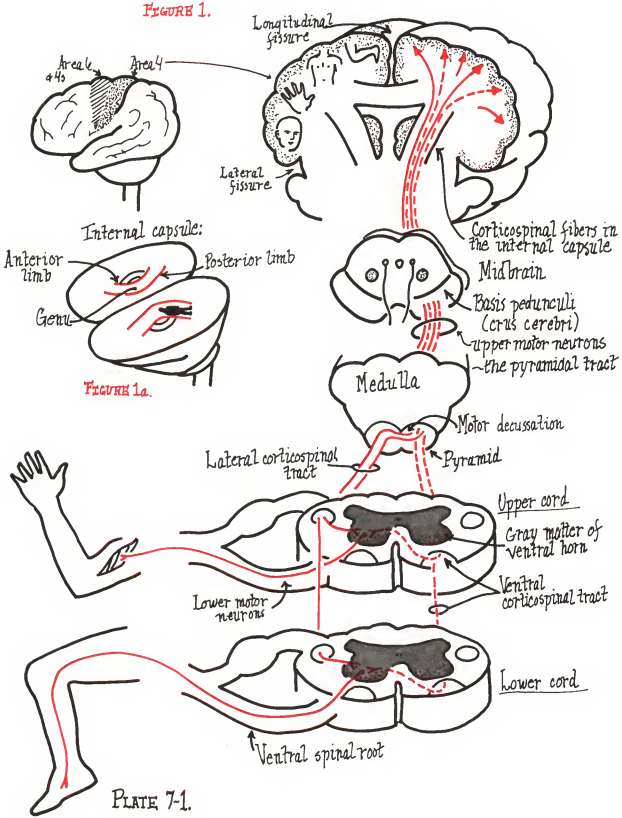


FIGURE 1.
Longitudinal fissure
Area 6
4 4b
Area 4
Lateral fissure
Internal capsule:
Anterior limb
Posterior limb
Genu
FIGURE 1a.
Corticospinal fibers in the internal capsule
Midbrain
Basis pedunculi (crus cerebri)
Upper motor neurons
The pyramidal tract
Medulla
Motor decussation
Lateral corticospinal tract
Pyramid
Upper cord
Gray matter of ventral horn
Ventral corticospinal tract
Lower motor neurons
Lower cord
Ventral spinal root
PLATE 7-1.

that supplies the muscles of the hand is disproportionately large, reflecting the great number of neurons needed to carry out such fine and complicated movements as violin playing, surgery, and writing. This localization is also seen in the internal capsule, the main cerebral passageway for ascending and descending fiber tracts. In a horizontal section (Figure 1a) of the cerebral hemisphere one sees that the internal capsule consists of an anterior limb, a posterior limb, and a connecting area between them, called the *genu.* The fibers that supply the face are situated in the genu, and those supplying the rest of the body are found in the anterior two-thirds of the posterior limb. If the genu is damaged, then the muscles of the face are affected, but if the middle part of the posterior limb is injured, then the leg muscles will not get nervous supply.

Suppressor Part of the Pyramidal Tract

Not all the neurons of the pyramidal tract have their origins in the precentral gyrus. Many of them originate in areas 4s and 6, which lie just anterior to the precentral gyrus (Figure 1). Pyramidal tract fibers originating here do not initiate impulses to voluntary muscles, but act as inhibitors, suppressors, or "brakes" on the lower motor neurons and prevent them from overdischarging when responding reflexively to sensory stimuli. If for some reason the suppressor fibers are damaged, then the lower motor neurons are freed from their control and fire excessively in response to reflex stimuli or discharge spontaneously. These conditions are known as *hyperreflexion* and *spasticity,* respectively, and are discussed in the next section.

CLINICAL ASPECTS

Lower Motor Neuron Paralysis

The best examples of lower motor neuron paralysis are when a nerve to a muscle is cut or when the cell bodies of the ventral horn are destroyed by poliomyelitis virus, which selectively attacks them. In both cases the muscles are deprived of their immediate nerve supply; they are unable to contract and become soft, flabby, and atrophic—characteristics of a flaccid paralysis. Naturally, because the motor limb of the reflex arc is damaged, the muscles can't respond reflexively to sensory stimuli.

If the cell bodies are destroyed, as in polio, then the axons cannot regenerate and the paralysis is permanent. If, however, a nerve is cut, then the part of the axon attached to the cell body can regenerate and some of the functions can be restored (see Chapter 1).

Upper Motor Neuron Paralysis

Upper motor neuron paralysis occurs when there is damage to the corticospinal tract anywhere along its path: the cell bodies in the precentral gyrus or their descending axons in the internal capsule, brainstem, or spinal cord. The most common site of injury is in the cerebral hemisphere, before the decussation. Injury results most often when an artery becomes stopped up and the neurons, deprived of their oxygen supply, die, producing what is known as a cerebrovascular accident (CVA) or, in popular language, a *stroke.* If the site affected is above the motor decussation, then the signs and symptoms will be seen in the muscles on the opposite side of the body. If the injury is below the decussation, say, a cut in the left half of the spinal cord, then the ensuing paralysis will be on the same side as the damage. This type of paralysis is different from a lower motor neuron paralysis in a number of essential ways. First of all, the lower motor neurons are not affected and the reflex arc is complete, and thus reflexes can be elicited. Second, the suppressor fibers originating in areas 4s and 6 are knocked out, and their braking effect on the lower motor neurons is no longer effective. The lower motor neurons now overdischarge to stimuli or even fire spontaneously. Clinically, this hyperreflexia manifests itself as follows: when the wrist of a paralyzed arm is grasped firmly, there will be a series of rapid, strong, muscular contractions, known as *clonus.* When the lower motor neurons discharge spontaneously, the muscles contract strongly, a condition known as *spasticity.* This upper motor neuron paralysis is spastic, as opposed to lower motor neuron paralysis, which is flaccid. In upper motor neuron paralysis a characteristic and specific type of reflex—the *Babinski reflex*—can be elicited. When the sole of the foot of a healthy person is stroked in a heel-to-toe direction, the toes will curl. However, in a patient who has an upper motor neuron lesion, the toes will fan apart and the big toe will flex dorsally. The exact route and mechanism of the Babinski reflex are still not fully understood. (In a normal infant up to the age of 6 months or so, where the myelinization of the axons is not complete, a Babinski response can be elicited routinely.) Also, certain superficial reflexes, such as the abdominal and cremasteric, which are elicited

when the skin is stroked, are lost. Once again the exact reason and mechanism are not clear.

A person who is "paralyzed" on one side of the body can frequently make crude movements of the trunk musculature on the affected side. The explanation for this is as follows: It is known that some of the lateral corticospinal fibers do not cross over at all, and it is believed that these uncrossed fibers, along with some of the crossed ones, supply the muscles of the trunk. Thus the trunk muscles of each side receive axons from both the right and left cerebral cortex. This arrangement is known as *bilateral innervation.*

Because a CVA may not destroy all the upper motor neuron axons to a group of muscles, those remaining intact can be utilized to regain some of the lost functions. This requires rehabilitation and involves such personnel as physiotherapists and occupational therapists.

Paralytic conditions may be defined according to the part(s) of the body affected: *Monoplegia* is paralysis of either an upper or a lower limb; *hemiplegia* is paralysis of an upper and a lower limb on the same side; *paraplegia* is paralysis of both lower limbs; *quadriplegia* is paralysis of all four limbs.

HISTORICAL NOTES

Hippocrates, one of the greatest physicians in history, noted over 2,000 years ago that injuries to one side of the head often produced paralysis on the contralateral (opposite) side of the body. Later Aretaeus of Cappadocia (circa 120–200 A.D.) said that this fact must be due to the nerves crossing somewhere in their pathway.

Chapter 8

PATHWAY TO VOLUNTARY MUSCLES OF THE HEAD

Our discussion of the pyramidal tract centered on those fibers that descend into the spinal cord and synapse there with lower motor neurons that go out and innervate voluntary muscles of the body. It did not include fibers to the voluntary muscles of the head because the lower motor neurons supplying them are not situated in the spinal nerves but are associated with cranial nerves originating in the brainstem. The basic framework, however, is the same as for the corticospinal tracts. It is a two-neuron pathway consisting of an upper motor neuron originating in the cerebral cortex whose axon descends and synapses with a lower motor neuron, which in turn goes out and stimulates voluntary muscles.

The cell bodies of the upper motor neurons are located in the lowest part of the precentral gyrus (motor cortex—area 4) adjacent to the lateral fissure (Figure 1). There is in addition another motor area for eyeball movements, which is situated in the middle frontal gyri (Figure 1a). Axons from here join descending fibers from the face area, and together they pass through the genu of the internal capsule. Because the fibers then enter the brainstem, or bulb, and terminate on lower motor neurons, they are called the *corticobulbar tract,* in contradistinction to the corticospinal tract. The cell bodies of the lower motor are concentrated in specific areas of the brainstem called *nuclei,* and their axons form many of the cranial nerves. These nerves differ from spinal nerves in that the sensory and motor fibers do not separate into dorsal and ventral roots. Furthermore, some cranial nerves have no sensory axons; all their fibers are lower motor neurons. To complicate the matter even further, there are cranial nerves that are entirely sensory in their makeup. Be that as it may, the cranial nerves that interest us here are those whose axons supply voluntary muscles. These are the oculomotor (III) and the trochlear (IV) nerves, whose nuclei are situated in the midbrain and whose axons go out to supply five of the six eyeball muscles and the levator palpebrae superioris; the trigeminal (V), the abducens (VI), and the facial (VII) nerves, all of which originate in the pons. The trigeminal nerve innervates the muscles of mastication as well as the anterior belly of the digastric, mylohyoid, tensor tympani, and tensor veli palatini muscles; the abducens supplies the last remaining eyeball muscle; the facial nerve, as its name implies, supplies all the muscles of facial expression. Finally, in the medulla are situated the nucleus of the glossopharyngeal nerve (IX), which innervates a single muscle in the pharynx (throat); the nucleus of the vagus nerve (X), which supplies muscles in the throat concerned with talking and swallowing (the nucleus of IX and X is really a single, common nucleus called the *nucleus ambiguus*); the nucleus of the hypoglossal nerve (XII), which supplies all the muscles of the tongue; and the accessory nerve (XI), which is an exception in that it doesn't supply muscles in the head but rather two very important ones in the neck—the sternomastoid and the trapezius.

No mention has yet been made of the crossing over of the corticobulbar tract because it isn't the same for all the cranial nerves just mentioned. The motor nuclei of all the cranial nerves mentioned, except VII and XII, receive innervation from both the right and left corticobulbar tracts; that is, each corticobulbar tract supplies both the right and left cranial nuclei (Figure 1). This bilateral innervation is a kind of biologic insurance. If the right tract is damaged, for example, the nuclei will still receive the upper motor neuron impulses from the intact left corticobulbar tract and there will be no impairment of muscle function.

The nuclei of cranial nerve XII, the hypoglossal nerve, receive only contralateral innervation; that is, the nucleus of the right side is supplied by

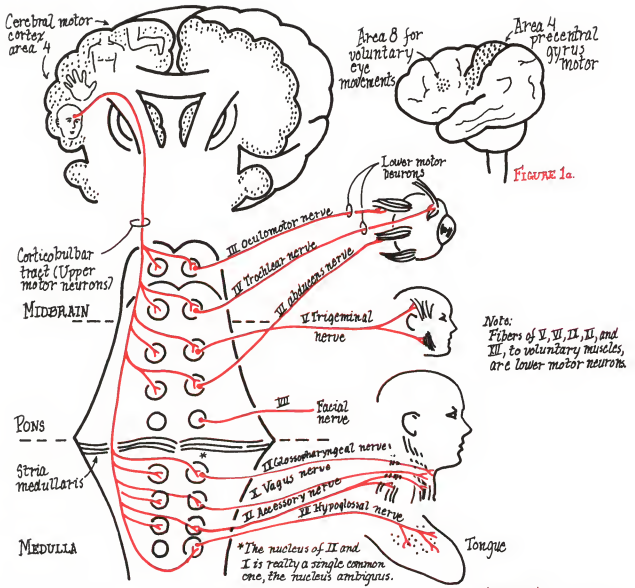


FIGURE 1a.

FIGURE 1.

FIGURE 2.

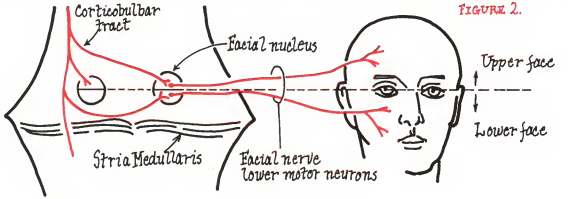


PLATE 8-1.

axons from the left corticobulbar tract and vice versa. The clinical implication is fairly obvious: a lesion to the left corticobulbar tract would result in loss of nerve supply to the right nucleus, and the muscles of the right side of the tongue would be paralyzed.

The facial nucleus, cranial nerve VII, combines features of both types of nuclei discussed so far. Its nucleus is divided into an upper part, which supplies the muscles of the upper half of the face, and a lower part, which supplies muscles in the lower half. The upper part of the nucleus receives bilateral innervation from the corticobulbar tract, whereas the lower part receives its supply from the contralateral tract (Figure 2).

CLINICAL ASPECTS

Upper Motor Neuron Lesion

As we have just seen, all the cranial motor nuclei (except the lower part of the facial and hypoglossal nerves) receive bilateral innervation. Therefore, if there is a lesion in one of the corticobulbar tracts, none of the nuclei or the muscles they supply would be affected. However, an upper motor neuron lesion (also known as a supranuclear lesion) would affect cranial nerve XII and/or the lower part of VII. If corticobulbar fibers to the facial nucleus are damaged, there is paralysis of the lower part of the facial muscles on the side opposite the lesion (Figure 2). The paralysis is spastic, and reflexes are present. Because the upper part of the facial muscles receives bilateral innervation, the patient can still move the brow on the paralyzed side of the face (Figure 2). If corticobulbar neurons to the hypoglossal nucleus are destroyed, the tongue muscles on the contralateral side will be paralyzed but will not atrophy (Figure 1). When the patient is asked to protrude the tongue, the muscles on the unaffected side cause it to deviate to the side on which the muscles are paralyzed.

Lower Motor Neuron Lesions

Lower motor neuron lesions are discussed in Chapter 13.

Chapter 9

SUBCORTICAL MOTOR AREAS

In lower forms of animals, such as sharks and birds, which do not have a cerebral motor cortex, movement is initiated by a group of nuclei, the basal ganglia, together with other subcortical areas. Such movement is highly coordinated and often very quick, but it is instinctive and crude. In humans there has been added to this old motor system a new, "higher" one—the cerebral motor cortex—which enables us to perform exceptionally skilled and purposeful movements, especially with the hands. This new system is called the pyramidal system, whereas the older, cruder one is the extrapyramidal system. For a while it was thought that the two were independent of each other, but now it is known that they are interconnected. Our knowledge of the old system is very incomplete, and a lot of what we think today may have to be modified by new discoveries tomorrow. With respect to terminology, there has been a tendency recently to use terms other than pyramidal and extrapyramidal, but this change in semantics hasn't been accompanied by any great increase in understanding. Also, many of the nuclei are grouped together and given special names (e.g., corpus striatum, lentiform nuclei). Because different authors don't always mean the same thing by the same term, in this book the nuclei and areas will be named individually.

Deep in the cerebral hemispheres are three well defined nuclei—the *caudate nucleus,* which lies medial to the anterior limb of the internal capsule, and the *globus pallidus* and *putamen,* which lie lateral to the genu (Figure 1; see also Appendix III, Figure 3). These three plus the amygdala constitute the basal ganglia. (The author has never been able to find out why the amygdala, which is concerned with olfactory reflexes, is considered a basal ganglion.) In the diencephalon is located the subthalamic nucleus of Luys, while in the midbrain are the red nucleus, the substantia nigra, and the reticular formation (Figure 2). All the above-mentioned structures make up the subcortical or primitive motor areas.

Various areas of the cerebral motor cortex, including areas 4, 4s, and 6, send fibers to the caudate, putamen, and pallidus (Figure 3). The globus pallidus, which also receives fibers from the caudate and putamen, is the main discharge center and is therefore connected with the subthalamic nucleus, substantia nigra, reticular formation, and red nucleus (Figure 3). In addition, the subthalamic nucleus and substantia nigra are connected to the reticular formation and red nucleus, which discharge to the lower motor neurons at all levels of the cord via the reticulospinal and rubrospinal tracts (Figure 3). Thus, there is, as was so aptly described by the neuroanatomist Elliot, a "cascading effect" with respect to nuclei and their discharges. Finally, the globus pallidus is connected to the thalamus by two tracts, the *ansa lenticularis* and the *lenticular fasciculus.* As they enter the thalamus these two tracts merge to form the *thalamic fasciculus.* The thalamus in turn is connected back to the caudate and areas 4, 4s, and 6, thus establishing a feedback mechanism. If our knowledge of the interconnections between different subcortical nuclei and their relationship to areas 4, 4s, and 6 is poor, then our understanding of how they operate and regulate motor activity is almost nil.

CLINICAL ASPECTS

Lesions in the primitive subcortical nuclei produce several diseases characterized by disturbances of muscle tone and various abnormal involuntary movements (dyskinesia). The most common and best known of these is *Parkinsonism.* Clinically, one sees a great increase in muscle tonus, leading to rigidity and slowness of movement. Combined with this is tremor, seen especially in the arms and hands, where it manifests itself in a characteristic pill-rolling motion. This tremor is most evident when the patient isn't doing anything with the hands—a resting tremor—but often disappears during purposeful movements. During walking, the head and shoulders are stooped, the gait is short and shuffling, and

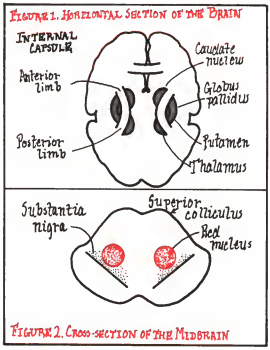


FIGURE 1. HORIZONTAL SECTION OF THE BRAIN

FIGURE 2. CROSS-SECTION OF THE MIDBRAIN

FIGURE 3.

PLATE 9-1.

there is a loss of automatic movements such as swinging of the arms. The face loses all signs of expression and becomes masklike. The cause of the disease is unknown, but at autopsy one most often sees degeneration of the globus pallidus and/or the substantia nigra. Parkinsonism may be seen in other conditions, such as Wilson's disease, in which there is abnormal copper metabolism that results in copper deposition in the globus pallidus, putamen, and liver. This causes their degeneration and the production of the above-mentioned symptoms. In mental patients high doses of chlorpromazine often produce signs of Parkinsonism as a temporary and unpleasant side effect.

Huntington's chorea is a condition characterized by rapid, jerky, nonrhythmic, involuntary movements of the extremities, trunk, and/or face. In contrast to Huntington's chorea, *athetosis* is a disease characterized by slow, bizarre, twisting movements, especially in the arms and fingers. In these two conditions the lesion is not found in a specific subcortical nucleus (i.e., it may be in the caudate or putamen or globus pallidus). Lastly, there is *hemiballism*, which is caused by a lesion in the subthalamic nucleus. In this disease there is a violent swinging motion of the arm or leg. The causes of all three diseases are still unknown. Tragically, there is as yet no cure or relief for these sufferers, and the movements cease only in sleep.

In animals, even those high on the evolutionary ladder such as chimpanzees, one can destroy experimentally the basal ganglia and no adverse signs are seen. This is very unfortunate—for humans but not for the monkeys—because it greatly limits the scope of research that can be done to find a cause or prevent or cure these afflictions.

However, experimental work does continue, and it has been shown that dopamine is an essential neurotransmitter in the basal ganglia. It is produced in the substantia nigra and passes up fibers to be used in the basal ganglia (Figure 3). In most cases of Parkinson's disease one sees that the substantia nigra is destroyed, and as a result there is a decrease in the amount of dopamine reaching the basal ganglia, thus producing the signs and symptoms of the illness. On the basis of this theory rests one of the current methods of treatment—that of supplying the basal ganglia with the missing dopamine. Dopamine cannot be given directly, however, because it does not pass the blood-brain barrier (see Chapter 1). Therefore, L-dopa, a necessary precursor of dopamine, is given because it can pass the blood-brain barrier and is then synthesized into dopamine. One often sees great improvement in patients following the administration of L-dopa.

Chapter 10

THE VESTIBULAR SYSTEM

It happens to all of us—suddenly, for one reason or another, one loses one's balance, starts to fall, and immediately a reflex reaction known as the "righting mechanism" comes into play in an attempt to regain equilibrium. This sense of loss of equilibrium and the reflex mechanisms to regain and maintain it are the function of the vestibular division of the eighth cranial nerve, the acoustovestibular nerve. The vestibular system is considered part of the extrapyramidal network because it does not involve the cerebral motor cortex and its actions are reflexive.

The receptor organ is located in the inner ear and consists of two fluid-filled sacs, the *utricle* and the *sacculus,* and three fluid-filled semicircular canals lying perpendicular to each other, which represent the three spatial planes (Figure 1). The fluid is *endolymph,* and suspended in it are specialized receptor cells—the *hair cells*—which are sensitive to fluid currents. When there is a shift or change of position of the head, the endolymph is set in motion. It stimulates the receptors, which transmit this information to the brain, which in turn sets off the appropriate reflex responses.

From the inner ear, primary neurons pass to the brain, with their cell bodies aggregated in the vestibular ganglion. Axons leave this ganglion and enter the brainstem, where they terminate in four vestibular nuclei situated in the area acoustica of the floor of the fourth ventricle (Figure 1). These nuclei have five major connections, which are discussed below one by one.

VESTIBULOCEREBELLAR CONNECTIONS

The cerebellum is the coordination center for motor activity and equilibrium. Therefore, from the superior and lateral vestibular nuclei, second-order (secondary) neurons pass up into the cerebellum via its inferior peduncle and terminate in the flocculonodular lobe (Figure 1). In addition, there are a few first-order axons that do not end in the vestibular nuclei but pass directly to the floccular nodulus (Figure 1). This then discharges back to the vestibular nuclei of *both sides* via the fastigial nucleus and the inferior peduncle. Thus, a cerebellar-vestibular feedback mechanism is established (Figure 1).

VESTIBULOSPINAL TRACTS

From the lateral vestibular nucleus, secondary neurons descend in the ipsilateral ventral white column and end by synapsing on lower motor neurons. These second-order neurons, which discharge reflexively to maintain equilibrium, form the lateral vestibulospinal tract (Figure 2). ("Lateral" here refers not to the fact that it originates in the lateral nucleus, but to the fact that it lies lateral to the medial vestibulospinal tract, which is discussed next.)

From the medial, superior, and inferior vestibular nuclei, second-order crossed and uncrossed neurons descend in the ventral white columns and terminate on lower motor neurons. These secondary neurons, which form the medial vestibulospinal tracts, also discharge reflexively to maintain body equilibrium (Figure 2).

VESTIBULO-OCULAR CONNECTIONS

Besides helping to maintain body equilibrium, the vestibular system also has the function of regulating eyeball movements in certain cases. For example, if one looks straight ahead and fixes one's eyes on an object and then turns one's head to the side, the appropriate eyeball muscles must contract in order for the eyes to remain "locked in" on the object. The regulation or control of this contraction is a function of the vestibular system. It

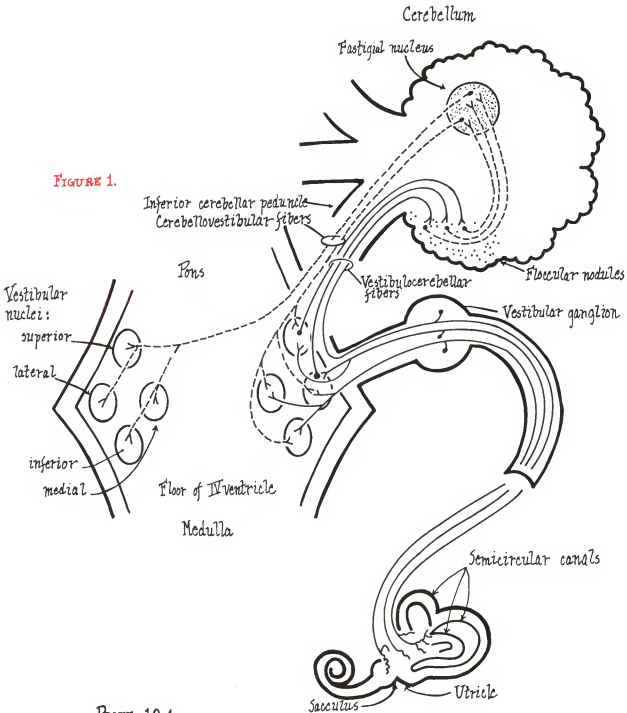


FIGURE 1.

PLATE 10-1.

works as follows: When one turns one's head, the endolymph in the semicircular canals, sacculus, and utricle is set in motion and stimulates the hair cells. This stimulus passes via the nerve and vestibular ganglion to the vestibular nuclei. We just mentioned that, from the medial, superior, and inferior nuclei, crossed and uncrossed neurons descend as the medial vestibulospinal tract. Just before descending, these neurons branch and give off axons that ascend in the pons and midbrain, where they synapse in the sixth (abducens), fourth (trochlear), and third (oculomotor) nuclei, which are all concerned with eyeball muscle movement. These ascending axons regulate the amount of eyeball muscle contraction, and form the medial longitudinal fasciculus (the MLF; Figure 2). (Some neuroanatomy books refer to the lateral vestibulospinal tract as the vestibulospinal tract, whereas the medial vestibulospinal tract is called the medial longitudinal fasciculus, or MLF.)

VESTIBULOCORTICAL CONNECTIONS

We can all sense a loss of equilibrium, or dizziness, if we are spun around quickly. This sensation implies vestibular connections to the thalamus, cerebral cortex, and consciousness. However, until now no such connections have been demonstrated morphologically. Some evidence has been obtained from electrophysiologic studies, but the problem remains unsolved.

ACCESSORY PATHWAY

We mentioned above that the fastigial nucleus of the cerebellum is part of the feedback mechanism to the vestibular nuclei, which then discharge to the lower motor neurons via the lateral and medial vestibulospinal tracts. There is, in addition, another pathway to maintain equilibrium. The fastigial nucleus is connected to the descending reticular areas and nuclei of the brainstem, which then discharge to the lower motor neurons via the multisynaptic reticulospinal tract (see Figure 3 in Chapter 11, and also Chapter 18, on the reticular systems).

CLINICAL ASPECTS

Lesions of the vestibular system often produce disturbances in equilibrium and walking straight. Because this system is connected with eyeball movements, lesions in it may also produce abnormal to-and-fro movements of the eyes, known as *nystagmus*. In nystagmus the eyes are constantly moving. First they move to one side as far as they can go, and then they snap back very quickly, then again they move slowly, and so on. There is thus a slow movement to one side and a quick one to the other; the nystagmus is called left or right nystagmus according to the direction of the quick movement. Most nystagmi are horizontal in direction, but there can also be vertical nystagmus. Nystagmus is very often seen in albinos.

Normal nystagmus can be seen in persons riding on trains. While they are looking out the window, their eyes will automatically focus on an object, follow it slowly until it is out of sight, and then snap back quickly and focus on another object. This slow-fast pattern of movement is repeated. Nystagmus is a complex phenomenon, and an excellent discussion of it, as well as of the vestibular system, can be found in Chapter 13 of *A Functional Approach to Neuroanatomy*, by House and Pansky (McGraw-Hill, 1960).

Another common symptom of vestibular injury is dizziness, although other conditions can also produce it. *Ménière's disease* is a disease of unknown etiology in which the patient suffers attacks of dizziness, ringing in the ears (tinnitus), and deafness.

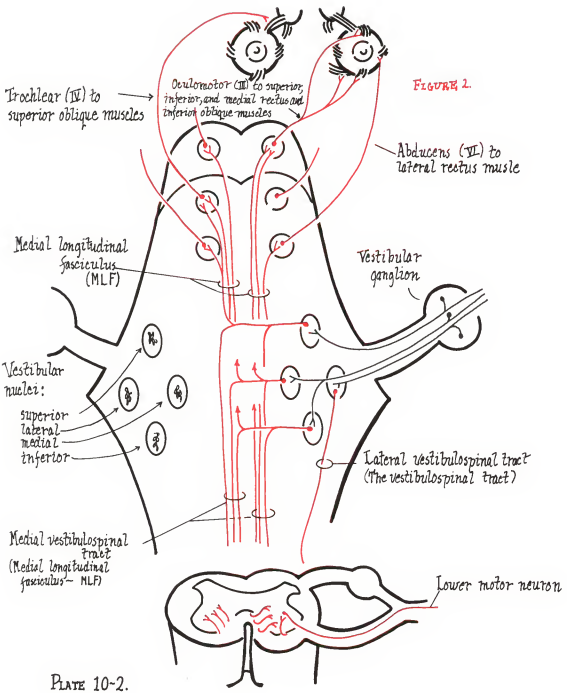


FIGURE 2.

PLATE 10-2.

Chapter 11

THE CEREBELLUM AND ITS PATHWAYS

The cerebellum (see Appendix III, Figure 4) is the control center for the coordination of voluntary muscle activity, equilibrium, and muscle tonus. It does not initiate movement; therefore, a person who has cerebellar injury does not become paralyzed. Rather, his or her movements are slow, clumsy, tremulous, and uncoordinated. The muscles may be hypertonic or hypotonic, and the person is unable to walk steadily but tends to sway, stagger, and fall. To carry out its three important functions, the cerebellum needs to receive a steady stream of information concerning:

1. The position and state of the muscles and joints, and the amount of tonus present
2. The equilibrium state of the body
3. What "orders" are being sent to the muscles from the cerebral motor cortex.

Receiving these three information "inputs," the cerebellum is then able to integrate them and, by means of "feedback" pathways, to regulate and control motor activity, equilibrium, and muscle tonus automatically and at an unconscious level. The discussion in this chapter considers each of the information inputs separately and then presents the feedback pathways.

THE SPINOCEREBELLAR PATHWAYS

Information concerning the condition of the muscles, the amount of tonus, and the position of the body is supplied by unconscious proprioceptive fibers, whose receptors are found in joints, tendons, and muscles. The cell bodies of these neurons are situated in the dorsal root ganglion, and the axons pass into the cord, from which they can reach the cerebellum by either one of two tracts. Most of those from the lower part of the body enter the dorsal horn, where they synapse with second-order neurons (Figure 1). Some of these secondary neurons ascend on the same side, in the ventral spinocerebellar tract of the lateral columns, and enter the cerebellum through its superior peduncle. The remaining secondary axons cross over to the contralateral side, enter the ventral spinocerebellar tract there, and ascend to the cerebellum. However, before passing into the superior cerebellar peduncle they cross back to the side from which they started (Figure 1).

Proprioceptive fibers from the upper part of the body mainly use the dorsal spinocerebellar tract. Here primary neurons synapse with secondary ones in Clarke's nucleus (Figure 1), which is found only in the upper part of the spinal cord (C_8–L_2). Secondary axons pass into the lateral columns on the same side to form the dorsal spinocerebellar tract, which enters the cerebellum through the inferior cerebellar peduncle. The important thing to remember is that all spinocerebellar fibers enter the cerebellum on the same side that they entered the cord.

The dorsal and ventral spinocerebellar tracts are the main bundles supplying proprioceptive impulses to the cerebellum. There are, however, a number of others, such as the trigeminocerebellar tract, from the muscles of mastication and the mandibular joint, and the olivocerebellar tract, as well as the reticulocerebellar and arcuocerebellar tracts.

VESTIBULOCEREBELLAR TRACT

From the superior and lateral vestibular nuclei arise the fibers that supply information concerning the equilibrium state of the body. They enter through the ipsilateral (homolateral) inferior peduncle and pass to the cerebellar cortex, especially that of the flocculus (Figure 2). Phylogenetically, the flocculus is the oldest part of the cerebellum and a center for equilibrium.

CORTICOPONTOCEREBELLAR TRACTS

When the cerebral motor cortex discharges to the lower motor neurons, the cerebellum must receive

FIGURE 1.

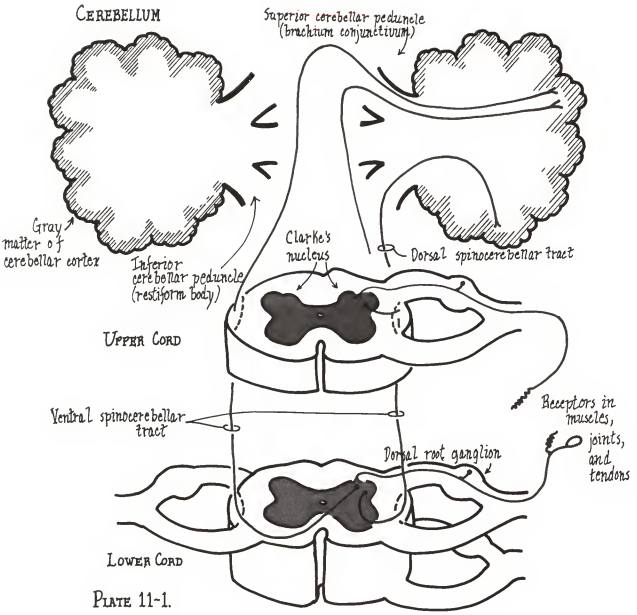


PLATE 11-1.

information about the nature of the discharge—to what muscles it is going, how strong it is, and so on—and it gets this information through the corticopontocerebellar tracts. The fibers originate in the cerebral cortex, descend through the internal capsule, and, at the level of the pons, synapse with second-order neurons in the pontine nuclei (Figure 2). The secondary axons now cross over to the other side and enter the cerebellum through its middle peduncle.

FEEDBACK PATHWAYS

The cerebellum, having received information concerning muscle states, tonus, and equilibrium, as well as the nature of the motor discharge to the muscles, integrates all this input (how, we don't know) and exerts its control via the following pathways:

From the cerebellar cortex, short neurons pass to several cerebellar nuclei—the emboliform, fastigial, globose, and dentate nuclei. The last-named nucleus is the most important; it sends out fibers through the superior peduncle that decussate and then enter the red nucleus of the midbrain (Figure 3). Not surprisingly, they are called the *dentorubro fibers,* or the dentatorubrothalamic tract, since some of them bypass the red nucleus and go up to the thalamus. The red nucleus can discharge up to the thalamus, which relays the information to the cerebral motor cortex; thus the feedback circuit is completed (Figure 3). The red nucleus can also discharge down to the lower motor neuron by means of the rubrospinal tract (Figure 3), and thus can influence the corticospinal impulses at the spinal level.

The cerebellum also discharges back, directly or through the fastigial nucleus, to the vestibular nuclei. These in turn relay the stimuli to the lower motor neurons by means of the vestibulospinal tract (Figure 3).

Finally, the cerebellum can influence the lower motor neurons by discharging to the reticular area and nuclei of the pons, midbrain, and medulla, which relay the discharge by the lateral and medial reticulospinal tract (Figure 3).

CLINICAL ASPECTS

Lesions of the cerebellum or its afferent and efferent tracts produce several characteristic signs, usually on the same side of the body as the injury:

1. *Asynergia* is the loss of coordination in performing motor acts. One sees decomposition of movement; that is, it is done in jerky stages instead of smoothly.
2. *Dysmetria* is the inability to judge distance and to stop movement at a chosen spot. Thus, in reaching for an object, a patient's hand will over- or underreach it. When asked to touch the tip of the nose, the patient's finger will hit the cheek—a pass-pointing phenomenon.
3. *Adiadochokinesia* is the inability to perform rapidly alternating movements, such as pronation and supination of the hands.
4. *Intention tremor* occurs during a movement and not at rest. In Parkinson's disease one sees just the opposite—a resting tremor.
5. *Abnormal gait*—The patient staggers and reels. To compensate, the patient walks with the feet spread apart.
6. *Falling*—The patient has a tendency to fall, especially to the injured side.
7. *Hypotonia*—The muscles are floppy and weak, but may be hypertonic in some cases.
8. *Dysphonia* is a slurred, explosive speech.
9. *Nystagmus* may be present.

Not all these signs or symptoms are present in every case of cerebellar damage. To test, ask the patient to perform the various movements described above, and check for their presence or absence.

FIGURE 2.

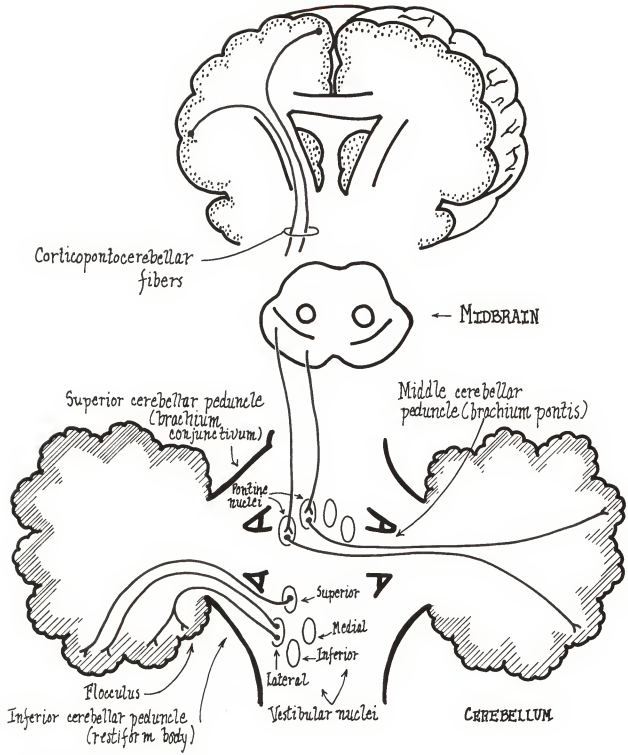


PLATE 11-2.

FIGURE 3.

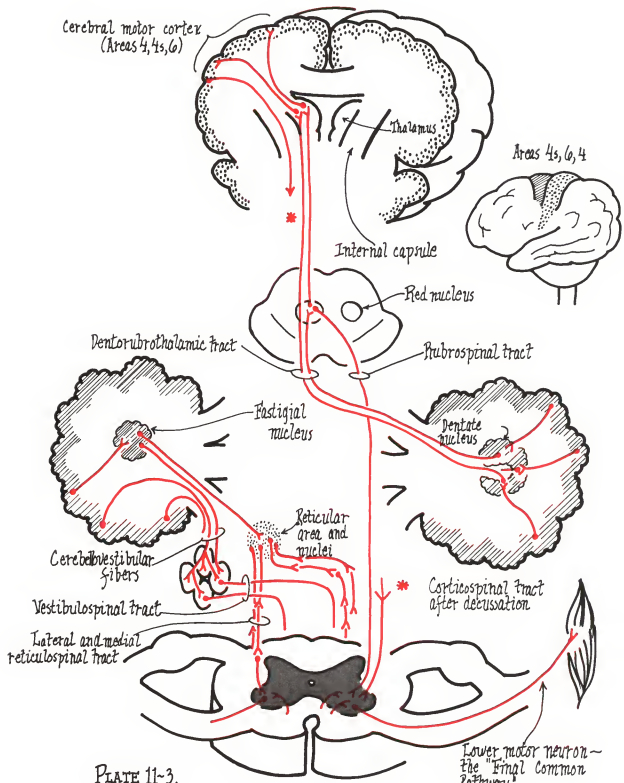


PLATE 11-3.

Chapter 12

THE AUTONOMIC NERVOUS SYSTEM

The autonomic nervous system (ANS), which is also known as the visceral or vegetative nervous system, stimulates and controls structures not under conscious control. If, for example, you are suddenly told that you're getting a surprise exam, your heart rate will probably increase, your mouth will go dry, you'll get "butterflies" in your stomach, and you'll start sweating—all automatic reactions to a stress situation. The autonomic nervous system stimulates three types of tissues: cardiac muscle, most glands, and all smooth muscle (that found in many organs and structures). The autonomic system is divided into two parts: the *sympathetic* nervous system and the *parasympathetic* nervous system, both of which supply (with two or three exceptions) the same organs and structures. However, the two systems are antagonistic to each other. For example, sympathetic stimulation of the heart results in an increased pulse rate, whereas parasympathetic stimulation slows it down. Sympathetic discharge results in dilation of the pupils, whereas parasympathetic stimulation produces constriction. The two systems are constantly discharging to the structures they supply, but there is a balance between them (Figure 1a). *This balance can be changed in either of two ways: by increasing the amount of stimulus in one part of the system* (Figure 1b), *or by decreasing the amount of discharge in the other* (Figure 1c). This very important principle forms the basis of much of neuropharmacology and is discussed later in greater detail.

THE SYMPATHETIC NERVOUS SYSTEM

The sympathetic nervous system is the one that dominates when a person is in a stress situation, be it physical or psychological. In both instances one feels threatened, and the body automatically reacts by preparing for "fight or flight." In these conditions the muscles will work harder, will need more oxygen, and will use more energy. Therefore, the bronchioles open up for quicker and greater passage of air; the heart beats stronger and faster; the arteries to the heart and voluntary muscles dilate, thereby bringing more blood to them; the arteries to the skin and peripheral areas of the body constrict, thereby shunting more blood to the active muscles (as a result the skin feels cold); the liver secretes glycogen for quick supply of energy; peristalsis slows down, since the body has no energy or time for digestion; the pupils dilate to get a better view of the surroundings; the hair "stands on end"; and one sweats. The last two responses are interesting evolutionary carryovers of more primitive defense reactions. The hairs of a cat that is threatened by a dog stand up, so that if the dog attempts to bite the body it gets a mouthful of hairs instead. As for sweating, did you ever try to grab and hold a person who is wet and slippery?

The sympathetic nervous system is based on a two-neuron pathway. The cell bodies of the first neurons are located in the lateral gray horn of the spinal cord, which is situated only between the first thoracic and the third lumbar (T_1–L_3) segments (Figure 2; the system is also called the thoracolumbar outflow). The axons leave the cord via the ventral roots and enter the sympathetic trunk. The sympathetic trunk is a series of ganglia and axon fibers on each side of the vertebral column that extends from the neck to the sacrum. It is also referred to as the paravertebral chain ganglia or the sympathetic chain. The question is: How do the primary axons that exit at T_1 reach the glands and smooth-muscled structures up in the head? After entering the sympathetic trunk, the axons ascend until they reach the superior cervical ganglion in the upper region of the neck (Figure 2). Here they synapse with secondary neurons,

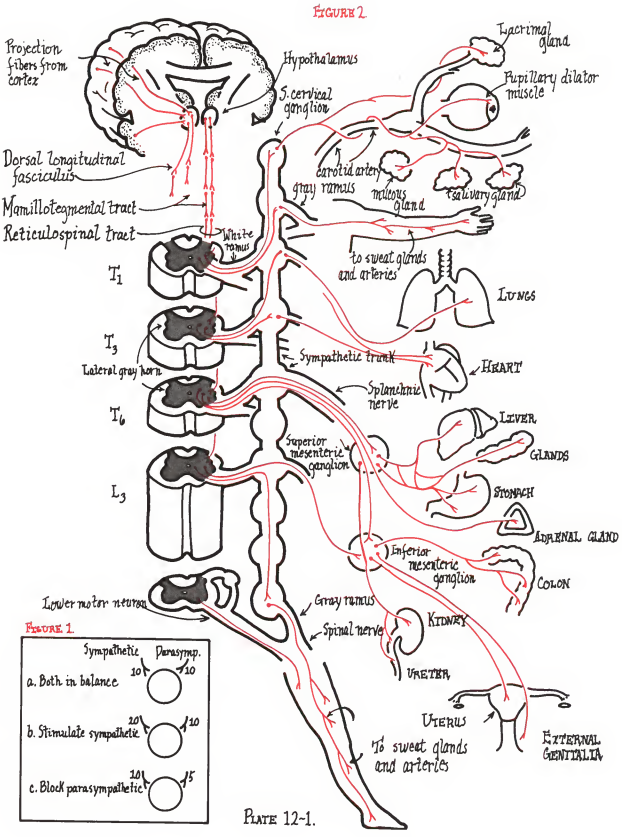


FIGURE 2.
Projection fibers from cortex
Hypothalamus
S. cervical ganglion
Lacrimal gland
Pupillary dilator muscle
Dorsal longitudinal fasciculus
Mamillotegmental tract
Reticulospinal tract
carotid artery
gray ramus
mucous gland
salivary gland
White ramus
T_1
to sweat glands and arteries
LUNGS
T_3
Lateral gray horn
Sympathetic trunk
HEART
Splanchnic nerve
T_6
LIVER
Superior mesenteric ganglion
GLANDS
L_3
STOMACH
ADRENAL GLAND
Inferior mesenteric ganglion
COLON
Lower motor neuron
Gray ramus
KIDNEY
Spinal nerve
URETER
URETUS
EXTERNAL GENITALIA
To sweat glands and arteries
FIGURE 1.
Sympathetic
Parasymp.
a. Both in balance
10
10
b. Stimulate sympathetic
20
10
c. Block parasympathetic
20
15
PLATE 12-1.

which go out and innervate the glands and other structures. The first neuron is called the *preganglionic neuron*, and its axon is myelinated; the second is the *postganglionic neuron*, and its axon is unmyelinated. This postganglionic axon reaches its destination by leaving the superior cervical ganglion and wrapping itself around the arteries that supply the innervated structures. It thus "hitches a ride" on the arteries until it comes to the glands and smooth-muscled structures, where it then peels off to innervate them (Figure 2).

Cell bodies of sympathetics destined to supply the heart and lungs are situated in the lateral gray horn of segments T_1–T_5. The axons leave the cord and enter the chain ganglia, where they synapse with postganglionic neurons. The axons of the latter leave the chain ganglia and form specific nerves that reach the heart and lungs (Figure 2).

Those sympathetics that supply the abdominal viscera are found in the lateral horn of T_5–T_{12}. Their axons enter the chain ganglia but do not synapse there. Rather, they pass through it and leave to form distinct nerves, the splanchnics, which terminate in the superior and inferior mesenteric ganglions of the abdomen. The postganglionic axons leave and form a netlike plexus that spreads out over the arteries to reach the various organs. Some of the preganglionic axons pass to the adrenal gland, the medullary cells of which are the postganglionic neurons that are specialized to secrete the hormone adrenaline (Figure 2).

Most of the preganglionic nerve cell bodies to the pelvic organs are located in the lateral horn of spinal segments L_1–L_2 (Figure 2). Their axons enter the sympathetic chain, pass through it without synapsing, and descend to end in the inferior mesenteric ganglion. From here the postganglionics fan out to supply the urinary and genital organs, as well as the descending and sigmoid colon and the rectum.

Accessory Details

The preganglionic sympathetic axons are myelinated and are therefore white. They leave the cord and peel off from the spinal nerve to form the white rami communicantes, which links up the spinal nerves with the sympathetic ganglia (Figure 2). The postganglionic axons are unmyelinated and therefore appear gray. Many rejoin the spinal nerves through the gray rami communicantes and pass out to supply the sweat glands and peripheral arteries of the head, upper extremity, the trunk, and the lower limbs (Figure 2).

The chemical transmitter between the postganglionic sympathetic axons and the structures they innervate is not acetylcholine, but adrenaline. If a patient is given a shot of adrenaline, the reaction is the same as if the sympathetic nervous system had discharged. Consequently, this sytem is also called the *adrenergic nervous system.* There are drugs that block the parasympathetic system, resulting in an imbalance between the two parts of the autonomic nervous system, and what one sees is similar in many ways to what happens when the sympathetics discharge (Figure 1c).

THE PARASYMPATHETIC NERVOUS SYSTEM

This system is also based on a two-neuron pathway, consisting of preganglionic and postganglionic neurons. However, there are great physiologic, anatomical, and pharmacologic differences between the two systems. Whereas the sympathetic nervous system is dominant in stress situations, the parasympathetic is most active when a person is relaxed and resting; the heart beat slows down, peristalsis and other digestive functions are active, and so on.

The chemical transmitter between the postganglionic parasympathetic axons and the structures they innervate is acetylcholine. Thus, if one gives a patient such a drug, the reaction resembles parasympathetic discharge.

As for the anatomy, the preganglionic cell bodies are located in the brainstem and in the gray matter of the cord in the sacral region. Thus another name for this system is the cranial-sacral outflow. In the brainstem the cell bodies are aggregated in several specific nuclei and the axons join cranial nerves III, VII, IX, and X. Being components of these nerves, they exit with them, pass out to the different regions, and, very near their destinations, enter specific named ganglia. Here the preganglionic axons synapse with short postganglionic fibers that innervate glands, the heart, and structures having smooth muscle. In many cases the ganglia are situated near, on, or within the structures innervated and the postganglionic fibers are microscopic.

At the level of the superior colliculus of the midbrain, preganglionic cell bodies are located in the *Edinger-Westphal nucleus* (Figure 3). The axons join the lower motor fibers of the oculomotor nerve (cranial nerve III), and together they leave the midbrain and course out to the eyeball (Figure 3). Near it, the preganglionic axons peel off and enter the ciliary ganglion, where they synapse with

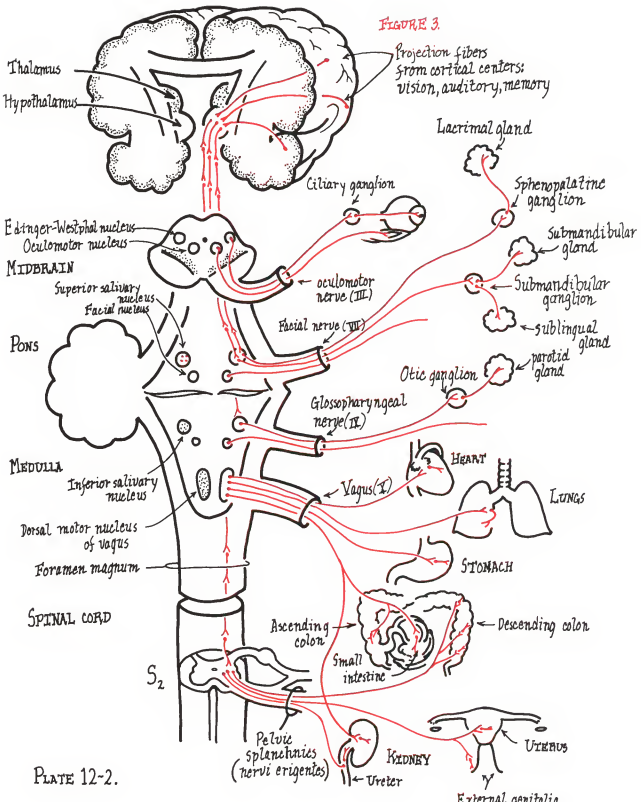


FIGURE 3.
Thalamus
Hypothalamus
Projection fibers from cortical centers: vision, auditory, memory
Lacrimal gland
Ciliary ganglion
Sphenopalatine ganglion
Edinger-Westphal nucleus
Oculomotor nucleus
MIDBRAIN
Submandibular gland
Superior salivary nucleus
Facial nucleus
oculomotor nerve (III)
Submandibular ganglion
Facial nerve (VII)
sublingual gland
PONS
Otic ganglion
parotid gland
Glossopharyngeal nerve (IX)
MEDULLA
HEART
Inferior salivary nucleus
Vagus (X)
LUNGS
Dorsal motor nucleus of vagus
STOMACH
Foramen magnum
SPINAL CORD
Ascending colon
Descending colon
Small intestine
S2
Pelvic splanchnics (nervi erigentes)
KIDNEY
URETHRA
Ureter
External genitalia

PLATE 12-2.

postganglionic neurons. These send out short axons to the pupillary constrictor muscle.

The preganglionic cell bodies associated with the facial nerve (cranial nerve VII) are situated in the superior salivary nucleus, and their axons pass out to the sphenopalatine (pterygopalatine) and submandibular (submaxillary) ganglia (Figure 3). From here the postganglionic axons course out to the lacrimal gland, as well as to the sublingual and submandibular glands.

As for cranial nerve IX, the glossopharyngeal, its preganglionic cell bodies are in the inferior salivatory nucleus and the axons go out to the otic ganglion, which sends out postganglionic fibers to the parotid gland (Figure 3).

The vagus nerve (cranial nerve X) is the most important cranial nerve because most of its fibers are parasympathetic neurons that innervate the heart, lungs, and all the abdominal viscera up to the left colic flexure. The preganglionic cell bodies are aggregated in the dorsal motor nucleus of the vagus, and the axons pass out and terminate in ganglia situated near or in the walls of the abovementioned organs (Figure 3). From these ganglia, postganglionic neurons innervate the structures.

The descending colon and the genital and urinary systems are supplied by the sacral outflow. Here the preganglionic cell bodies are situated in the lateral area of the gray matter of spinal segments S_2–S_4. The axons leave through the ventral roots and soon separate from the spinal nerve to form the pelvic splanchnic nerves or nervi erigentes, which reach the mural ganglia of the descending colon, ureter, and genital organs (Figure 3).

Accessory Detail

The hypothalamus is the control and integrative center for the autonomic nervous system, and its actions are automatic and not regularly subject to conscious control. (The hypothalamus is part of the diencephalon and lies below the thalamus on either side of the third ventricle (Figure 3).) It receives fiber bundles mainly from higher cortical centers, such as vision, auditory, personality, etc., and then discharges the appropriate impulses down the cord to the sympathetic or parasympathetic preganglionic neurons. It does this by means of the dorsal longitudinal fasciculus, the mamillotegmental tract, and the multisynaptic reticulospinal tract (see Chapters 17 and 18 on the reticular formation and the hypothalamus).

Pain pathways from the viscera are poorly understood, but it is generally accepted that the impulses travel via the autonomic nerves. Thus the system is afferent as well as efferent.

CLINICAL ASPECTS

Adrenergic drugs are those whose action mimics sympathetic activity. They are also known as sympathomimetics and are used primarily in hospitals to deal with cases of falling blood pressure and cardiac arrest. They are also used to dilate the bronchioles in asthma and in cases of anaphylactic shock. Sympathetic antagonists are those drugs which block sympathetic activity. In recent years they have become among the most important and widespread drugs, primarily in the treatment of hypertension.

Cholinergic (or parasympathomimetic) drugs are those whose action duplicates parasympathetic activity. These have very little use in medical practice. The parasympathetic blocking agents have a wider use and among the most common is atropine (belladonna*), which causes marked pupillary dilation and is therefore used by ophthalmologists when they want to take a good look at the eye.

*This drug got its name from the fact that, during the Renaissance, women used it to dilate their pupils in an attempt to make their eyes more beautiful (*bella donna* is Italian for "beautiful lady"), even though it seriously blurred their vision.

Chapter 13

CRANIAL NERVES

The 12 pairs of cranial nerves, which we have already discussed in passing in previous chapters, are considered in detail here. These nerves can be grouped in several ways, the first way being according to their central location (see figures in this chapter and also Appendix II). Cranial nerves I and II, the olfactory and optic nerves, are connected to the telencephalon and diencephalon, respectively. Nerves III and IV, the oculomotor and trochlear nerves, are connected with the midbrain; the trigeminal (V), the abducens (VI), and the facial (VII) nerves are located in the pons; the remaining nerves (VIII, IX, X, XI, and XII) are associated with the medulla. It is important to know this location plan: if a patient exhibits signs of a specific cranial nerve injury, then the site of the lesion can be pinpointed.

Another way to group cranial nerves is according to their functional neuronal components. Some have only sensory neurons; they are (Figure 1a):

I, the *olfactory* nerve, concerned with smell (see Chapter 16)

II, the *optic* nerve, which deals with vision (see Chapter 15)

VIII, the *acoustovestibular* nerve, concerned with hearing and equilibrium (see Chapters 10 and 14).

Other cranial nerves are composed only of motor neurons to voluntary muscles; they are (Figure 1b):

IV, the *trochlear* nerve, which innervates the superior oblique muscle of the eyeball. If the nerve or its nucleus is damaged, the muscle will be paralyzed and there will be difficulty in turning the affected eye downward and medially.

VI, the *abducens* nerve, which innervates the lateral rectus muscle of the eyeball. If this nerve or its nucleus is injured, the muscle becomes paralyzed and the patient can't turn the eye laterally. In time, the unopposed medial rectus causes the eye to be pulled medially, thus producing a medial strabismus (squint).

XI, the *accessory* nerve, which innervates two important muscles outside the head: the trapezius and the sternocleidomastoid muscles. These two neck muscles are also supplied by spinal nerves; thus, if the accessory nerve or its nucleus is damaged, the muscle will still function partially. However, the patient will have difficulty shrugging the shoulder on the affected side and turning the head to the opposite side.

XII, the *hypoglossal* nerve, which supplies all the muscles of the tongue. Again, if this nerve or its nucleus is damaged, then the muscles on the affected side become paralyzed, and the tongue, when protruded, will deviate to the paralyzed side. The reason for this is that tongue muscles are so arranged that, if one side is paralyzed, then, upon protrusion, the muscles on the unparalyzed side pull the tongue over to the paralyzed side.

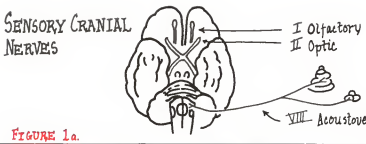


FIGURE 1a.

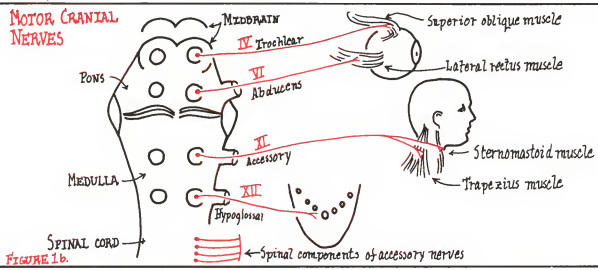


FIGURE 1b.

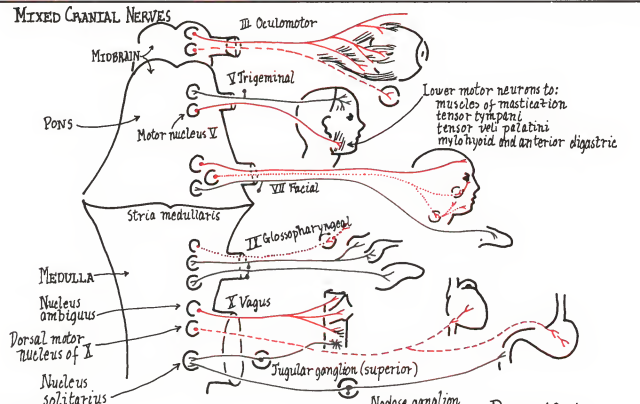
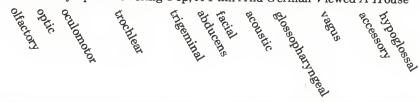


FIGURE 1c.

PLATE 13-1.

Note: a very old mnemonic device for use in naming the 12 cranial nerves is:
"*O*n *O*ld *O*lympus' *T*owering *T*op, *A F*inn *A*nd *G*erman *V*iewed *A H*ouse"



Modesty prevents presentation of the racier versions—ask the upperclassmen.

The remaining cranial nerves (III, V, VII, IX, and X) have mixed functional neuronal components (Figure 1c). Each of these mixed cranial nerves is discussed below in detail.

THE OCULOMOTOR NERVE (III)

The motor nucleus of the oculomotor nerve is located in the midbrain below the aqueduct of Sylvius at the level of the superior colliculus (Figure 2 and Appendix II, Plate X). From it emerge voluntary motor fibers (lower motor neurons), which leave the brainstem at the interpeduncular fossa and pass out to the orbit. Here they supply the following four eyeball muscles: the superior rectus, inferior rectus, and medial rectus muscles, and the inferior oblique muscle. In addition, they innervate the levator palpebrae superioris, which is responsible for lifting the upper eyelid.

The Edinger-Westphal nucleus is the parasympathetic nucleus of the oculomotor nerve and is situated just dorsal to the motor nucleus (Figure 2). Preganglionic fibers leave it, join the voluntary motor fibers, and pass out to the orbit. There the parasympathetic fibers separate, and most of them terminate in the ciliary ganglion (Figure 2). Here they synapse with postganglionic fibers that stimulate the sphincter pupillae muscle, causing the pupil to constrict. Other postganglionic fibers from the ciliary ganglion pass to the ciliary muscle, which is concerned with lens accommodation for near vision.

Clinical Aspects

Because the oculomotor nucleus receives a bilateral upper motor neuron supply via the corticobulbar tract (Chapter 8), one rarely sees an upper motor (a supranuclear) lesion that affects this nerve. However, if the oculomotor nerve is damaged, there is a lower motor neuron paralysis of the muscles it supplies, and the eyeball is pulled laterally and downward by the unopposed lateral rectus muscle (supplied by the abducens nerve) and the superior oblique muscle (supplied by the trochlear). Because the levator palpebrae is paralyzed, the upper eyelid droops—a condition known as *ptosis*. In addition, the parasympathetic fibers will be damaged and, as a result, the sphincter pupillae will be paralyzed. The dilator pupillae, supplied by the sympathetics, is now unopposed; consequently the pupil is widely dilated and cannot constrict (in other words, there is a "fixed pupil"). Also, oculomotor nerve damage causes difficulty in visual accommodation because the ciliary muscle is paralyzed.

THE TRIGEMINAL NERVE (V)

This trigeminal nerve has general sensory fibers as well as voluntary motor neurons. The sensory fibers (Chapter 6) convey general sensations of pain, temperature, touch, pressure, and proprioception from the face, cornea, mouth, nose sinuses, tongue, teeth, meninges, outer surface of the eardrum, and temporomandibular joint. The motor component consists of voluntary or lower motor neurons that supply the four muscles of mastication—the temporalis, the masseter, the lateral, and the medial pterygoids (Figure 1c). In addition, the trigeminal motor fibers innervate the anterior belly of the digastric, the mylohyoid, and the tensor tympani and tensor veli palatini muscles. The motor nucleus of the trigeminal nerve is located in the pons near the main sensory nucleus.

Clinical Aspects

If the entire nerve is cut or damaged, there will be a complete loss of sensation in the facial area on the same side, as well as a lower motor neuron lesion that produces difficulty in chewing and speaking. Because this nerve receives a bilateral innervation from the cerebral cortex, one rarely sees cases of upper motor neuron lesions (see also "Clinical Aspects" in Chapter 6).

THE FACIAL NERVE (VII)

The facial nerve is a more complex nerve that has three major components:

1. Special sensory fibers for taste from the anterior two-thirds of the tongue

FIGURE 2. OCULOMOTOR OR CRANIAL NERVE III

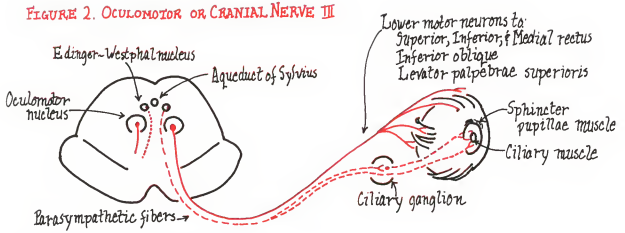


FIGURE 3. GLOSSOPHARYNGEAL OR CRANIAL NERVE IX

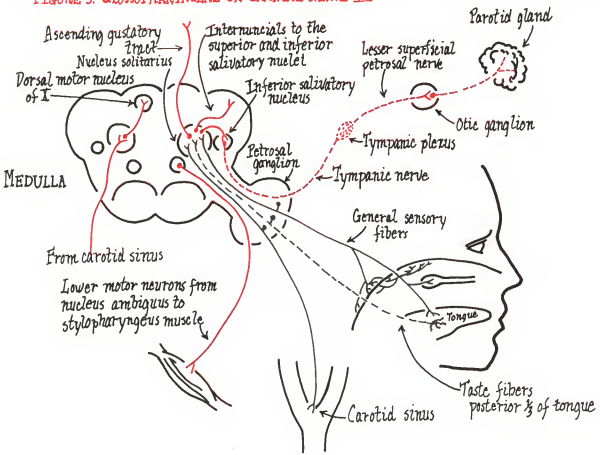


PLATE 13-2.

2. Parasympathetic fibers to the sublingual, submandibular (submaxillary), and lacrimal glands
3. Voluntary motor fibers to all the muscles of facial expression.

The taste receptors are located on the anterior two-thirds of the tongue, and their fibers pass back to the brainstem (Figure 4). In their course, they merge with the lingual branch of the trigeminal nerve, but then they separate from it to form the nerve known as the *chorda tympani.* This nerve enters the skull through a small fissure and passes into the temporal bone, in which is situated the geniculate ganglion. Here are located the cell bodies of the taste neurons, whose axons pass into the pons and end in the nucleus solitarius (Figures 4 and 4a). From this nucleus second-order ascending gustatory tracts arise which reach conscious levels; however, their exact course is unknown. In addition, there are reflex pathways for taste sensations. For example, when one tastes something pleasant there is a reflex salivation, and this pathway involves the parasympathetic component of the seventh as well as the ninth cranial nerve. From the nucleus solitarius internuncials pass down to the superior salivary nucleus and synapse with preganglionic neurons (Figure 4). Their axons leave the pons, enter the internal auditory meatus, and travel through the geniculate ganglion. They then separate from the rest of the facial nerve fibers to form the chorda tympani, which merges with the lingual nerve. After "hitching a ride" with the lingual nerve, the preganglionic parasympathetics again separate and terminate in the submandibular (submaxillary) ganglion. Here they synapse with the postganglionic neurons, which stimulate the submandibular and sublingual salivary glands. From the superior salivatory nucleus other preganglionic parasympathetic neurons follow a different course and reach the sphenopalatine (pterygopalatine) ganglion, where they synapse with postganglionic neurons (Figure 4). These postganglionic neurons follow a complicated pathway to reach the lacrimal gland and the mucus-secreting cells of the nose and mouth (Figure 4).

The last major component of the facial nerve is voluntary motor fibers to all the muscles of facial expression. Their nucleus is found in the tegmentum of the pons below the nucleus of cranial nerve VI (Figure 4). The emerging motor fibers pass up and loop around the abducens nucleus, causing a bulge on the floor of the fourth ventricle that is known as the *facial colliculus.* These motor fibers then join the rest of the components and enter the internal auditory meatus. After the taste and parasympathetic neurons have separated from the main bundle, the remaining voluntary motor fibers leave the skull at the stylomastoid foramen and separate into five main branches that supply all the muscles of facial expression, as well as the posterior belly of the digastric muscle. Within the temporal bone some motor fibers supply the stapedius muscle of the middle ear, which acts as a "brake" on the hearing apparatus and prevents hyperacusis (i.e., normal sounds heard abnormally loud on the affected side).

Clinical Aspects

One of the most common pathologic conditions involving the seventh cranial nerve is Bell's palsy. In this condition, nerve damage of unknown etiology results in a characteristic lower motor neuron paralysis of all the muscles of facial expression on the affected side. The person is unable to close the affected eye because the orbicularis oculi muscle is paralyzed. The unopposed muscles on the unaffected side contract and pull the mouth up in a characteristic grin. The lesions may affect the stapedius muscle, and the patient will suffer from hyperacusis. In addition, there may be a partial loss of taste and salivation, and a total loss of lacrimation on the affected side.

Because the lower part of the motor nucleus receives its upper motor neuron supply only from the contralateral corticobulbar tract (Chapter 8), an upper motor or supranuclear lesion will produce a contralateral spastic paralysis of the muscles of the upper half of the face. Because the muscles of the upper half of the face have a bilateral nerve supply, the patient with such a lesion can still close the eyes and wrinkle the brow. These two actions help differentiate between Bell's palsy, which is a lower motor neuron paralysis, and an upper motor neuron lesion.

THE GLOSSOPHARYNGEAL NERVE (IX)

The glossopharyngeal nerve also has three major components:

1. Special sensory taste neurons from the posterior third of the tongue
2. Parasympathetic fibers to the parotid gland
3. General sensory neurons from the auditory tube, the back of the tongue, the inner surface of the tympanic membrane, the pharynx, and the carotid sinus.

On the surface of the posterior third of the tongue are situated the taste receptors of the

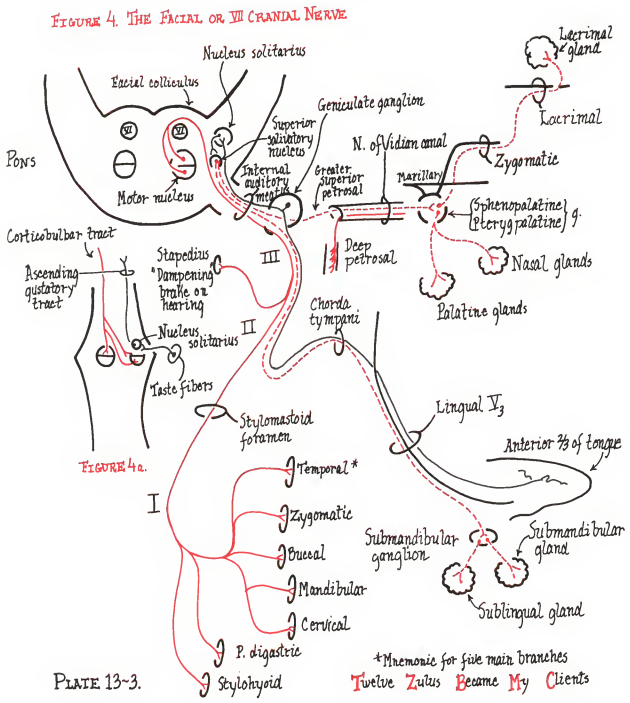


FIGURE 4. THE FACIAL OR VII CRANIAL NERVE
Nucleus solitarius
Facial colliculus
Geniculate ganglion
Superior salivatory nucleus
Internal auditory meatus
Greater superior petrosal
N. of Vidian canal
Lacrimal gland
Lacrimal
Zygomatic
Maxillary
(Sphenopalatine) (Pterygpalatine) g.
Nasal glands
Palatine glands
Deep petrosal
Chorda tympani
Lingual V3
Anterior 2/3 of tongue
Submandibular ganglion
Submandibular gland
Sublingual gland
Pons
Motor nucleus
Corticobulbar tract
Ascending gustatory tract
Stapedius
*Dampening brake on hearing
Nucleus solitarius
Taste Fibers
Stylomastoid foramen
FIGURE 4a.
Temporal*
Zygomatic
Buccal
Mandibular
Cervical
P. digastric
Stylohyoid
I
II
III
*Mnemonic for five main branches
Twelve Zulus Became My Clients
PLATE 13-3.

ninth cranial nerve. The cell bodies of these neurons are located in the petrosal ganglion (Figure 3), and their axons end in the nucleus solitarius, which extends down from the pons into the medulla. Here they synapse with cell bodies whose ascending gustatory fibers eventually reach conscious levels, but their pathway and final cortical localization are unclear. As is the case with the seventh nerve, there are also reflex arcs involving taste. From the solitarius nucleus, short internuncials pass to the inferior salivatory nucleus and synapse with preganglionic parasympathetic neurons. The axons of the latter leave the medulla along with the other glossopharyngeal fibers, but then separate and follow a long course (Figure 3) to reach the otic ganglion. Here they synapse with postganglionic parasympathetic neurons that stimulate the parotid salivary gland. Other internuncials from the nucleus solitarius pass up and synapse in the superior salivary nucleus, whose fibers reach the sublingual and submandibular glands (see the discussion of the facial nerve above).

The last major component of the glossopharyngeal nerve is the general sensory component, involving pain, pressure, touch, and temperature. The receptors are found in the Eustachian tube, the middle ear, the inner surface of the tympanic membrane, the uvula, the carotid sinus, and the nasal and oral pharynx. The fibers from these areas pass back to the inferior or petrosal ganglia, which houses their cell bodies, and terminate in the nucleus solitarius. Here they synapse with neurons of various tracts. Some of these ascend to consciousness, and others set off important reflexes, such as the gag reflex (see "Clinical Aspects"). Another example involves the carotid sinus, which is sensitive to blood pressure changes. A rise in blood pressure stimulates this receptor, which fires off a compensatory reflex. From the nucleus solitarius an internuncial neuron passes to the dorsal motor nucleus of the vagus nerve (cranial nerve X) and synapses with a parasympathetic neuron. The latter descends to the heart and stimulates it to slow down the heart rate, thus lowering the blood pressure.

The glossopharyngeal nerve also has voluntary motor neurons that stimulate the stylopharyngeus muscle (Figure 3).

Clinical Aspects

If the uvula or oral pharynx is touched, a gag or swallowing reflex is set off and the trachea is closed by the epiglottis. However, when a patient is under general gas anesthesia, this reflex does not work. Furthermore, the unconscious patient often vomits. It is therefore *absolutely imperative* that, before general surgery, *no food or liquid be given* to the patient for 12 hours before the procedure: otherwise the patient may vomit while unconscious and the acid contents from the stomach will enter the now wide-open trachea and lungs—with a fatal result.

THE VAGUS NERVE (X)

The vagus nerve, a vital nerve, has two major components:

1. Parasympathetic fibers to all the autonomic structures of the chest and abdomen up to the left colic flexure (Chapter 12, Figure 3)
2. Voluntary motor fibers to the muscles of larynx and pharynx involved in talking and swallowing.

The vagus also has general sensory fibers from the viscera, the carotid body (a chemoreceptor), the carotid sinus, the dura of the posterior cranial fossa, and the lower part of the pharynx. The cell bodies are located in the nodose or inferior ganglia, and the axons end in the nucleus solitarius (Figure 1c).

The parasympathetic fibers arise from the dorsal motor nucleus of the vagus nerve, which is found in the floor of the fourth ventricle of the medulla, just lateral to the hypoglossal nucleus (Appendix II, Plate XIV). The preganglionic fibers leave and descend into the chest and abdomen, where they synapse in ganglia that are situated on or in the organs that are innervated (Figure 1c of this chapter and Chapter 12, Figure 3). Finally, the vagus has general sensory fibers from a small area of the external ear; cell bodies from these are situated in the jugular or superior ganglion.

The motor fibers arise in the nucleus ambiguus of the medulla (Figure 1c and Appendix II, Plate XIV) and leave the brainstem with the parasympathetic fibers. They soon branch away from the vagus and supply all the muscles of the larynx and most of those in the pharymx. Damage to these motor fibers or their nucleus results in a lower motor neuron paralysis, with difficulty in talking (dysphonia) or swallowing (dysphagia).

Chapter 14

THE AUDITORY PATHWAY

The eighth cranial nerve, the acoustovestibular (vestibulocochlear), is entirely sensory and has two important parts: the acoustic part, which transmits sound impulses from the ear to the brain, and the vestibular part, which is concerned with maintaining body equilibrium. This chapter deals with the acoustic division, which is basically very simple.

In the cochlear apparatus of the inner ear (see Appendix III, Figure 7) are situated specialized receptors—the *hair cells*—which are stimulated by auditory vibrations from the external and middle ear. In the cochlea these hair cells synapse with primary neurons, the cell bodies of which are localized in the spiral ganglion, also situated in the cochlea (Figure 1). From here axons pass to the brainstem, enter it at the pontomedullary junction, and immediately bifurcate, with one branch terminating in the *dorsal cochlear nucleus* and the other in the *ventral cochlear nucleus* (Figure 1). From the dorsal cochlear nucleus some secondary axons cross over to the other side and ascend to reach the nucleus of the inferior colliculus. Others do not cross, but ascend ipsilaterally and also terminate in the nucleus of the inferior colliculus (Figure 1). These ascending crossed and uncrossed fibers comprise the *lateral lemniscus.*

Most of the axons from the ventral cochlear nucleus decussate and pass up in the lateral lemniscus to end in the nucleus of the inferior colliculus. A few do not cross over but ascend in the ipsilateral lateral lemniscus (Figure 1). Thus, both the dorsal and the ventral cochlear nuclei send out crossed and uncrossed fibers to the nucleus of the inferior colliculus. From here fibers are relayed out, via the brachium of the inferior colliculus, to the medial geniculate body, which lies adjacent to the superior colliculus. In this body they synapse with neurons, the axons of which form the auditory radiations that end in the cortex of the superior temporal gyrus, known as areas 41 and 42—the primary hearing center.

ACCESSORY DETAILS AND CLINICAL ASPECTS

1. The decussating axons from the dorsal and ventral cochlear nuclei form a large distinct mass, the *trapezoid body* (Figure 1).
2. The right and left nuclei of the inferior colliculus are connected to each other by commissural neurons (Figure 1).
3. Some of the fibers in the lateral lemniscus don't end in the nucleus of the inferior colliculus but pass straight up to the medial geniculate body (Figure 1).
4. On the other hand, many axons from the dorsal and ventral cochlear nuclei do not ascend directly to the midbrain, but rather make many synaptic stops along the way. For example, crossed fibers from both nuclei synapse in the superior olivary nucleus, which then relays up to the higher areas (Figure 1). This is not important clinically. What is important is the fact that each auditory cortex receives fibers from the left and right cochlear nuclei, or, put another way, the cochlear nuclei of the right side project onto the left and right auditory cortex, and left cochlear nuclei project to both hearing centers. The clinical significance of this bilateral representation is obvious. If, for example, the right auditory cortex is damaged, then the patient will still hear from both ears, using the intact left auditory cortex. This holds true for damage at other sites along the central pathway, namely, the right medial geniculate body, the right nucleus of the inferior colliculus, or the right lateral lemniscus. However, if the right auditory nerve is cut or damaged anywhere along its path—from the ear up to and including the cochlear nuclei—then the person will be deaf in the right ear, and what holds true for the right side is true for the left.
5. From the nucleus of the inferior colliculus, internuncial axons pass out to various motor centers to mediate auditory reflexes. For ex-

FIGURE 1

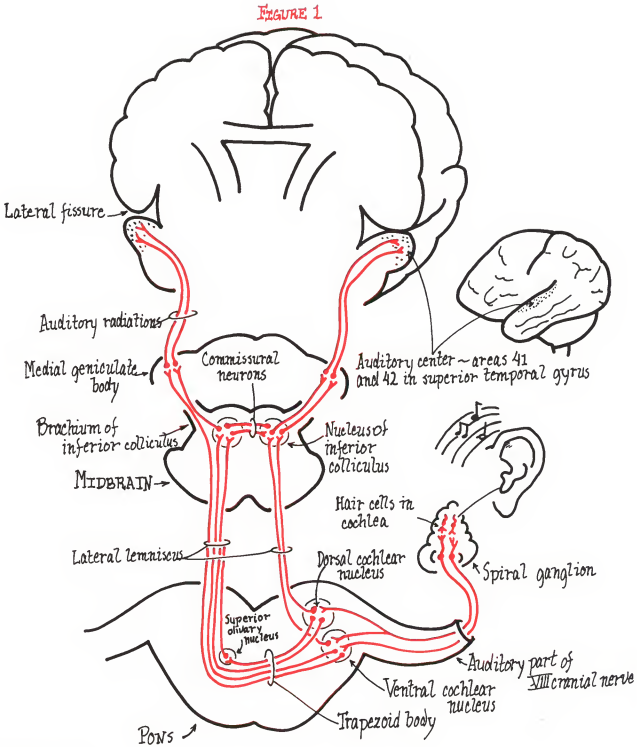


PLATE 14-1.

ample, when one hears a sudden loud noise, the eyes close and the body "jumps," both of which are reactions of the startle reflex.

In the United States, deafness is a widespread problem affecting millions of people. It is generally divided into two main groups. The first is *conduction deafness,* or middle ear deafness, in which a mechanical impediment prevents the sound from reaching the cochlea. The impediment may be a torn eardrum, blockage of the auditory canal, or other cause. The most common cause, however, is otosclerosis, in which the stapes of the middle ear becomes fixed in the cochlea and cannot transit its vibrations. The second group is *sensorineural deafness.* As its name implies, it is caused by damage to either the cochlea or cranial nerve VIII.

The distinction between these two groups is important from both a diagnostic as well as a treatment point of view. In conduction or middle ear deafness, a patient will hear poorly or not at all a vibrating tuning fork that is placed near the ear. However, the patient will hear the tuning fork if it is placed against the skull, because now the vibrations bypass the middle ear and are transmitted directly to the cochlea. On the other hand, in sensorineural deafness the patient will have difficulty in hearing a vibrating tuning fork both via air conduction and via bone conduction.

In recent years great progress has been made in the treatment of middle ear deafness, especially in otosclerosis. By means of microsurgery, the fixed stapes can be made mobile or even replaced, and the majority of patients show great improvement in their hearing.

There are many causes for sensorineural deafness, but the list below mentions only the most common:

1. Rubella infection in a pregnant woman very often causes her child to be born totally deaf.
2. Some antibiotics (e.g., streptomycin and neomycin) when given in large does may cause partial or total deafness, often accompanied by vestibular disturbances.
3. Atrophy of the cochlea is one of the most common causes of deafness in the aged.
4. Tumors such as acoustic neuroma of cranial nerve VIII can produce deafness.
5. There are many forms of hereditary deafness based on genetic defects.

Chapter 15

VISUAL PATHWAYS AND OPTIC REFLEXES

The visual pathways are among the more important pathways of the nervous system. Because injuries to them are common, the physician must know and understand them "cold."

Light rays from an object in the visual field enter the eyeball (Appendix III, Figure 6), are inverted by the lens, and strike the nervous layer, the *retina.* The retina is composed of several layers and types of neurons, among them being the light-sensitive *rods* and *cones.* Each eye has a temporal and a nasal visual field, and, because of the inversion by the lens, the temporal visual field is projected onto the nasal retinal field and the nasal visual field falls on the temporal retinal field (Figure 1). Loss of vision is always described with reference to the visual fields, not the retinal fields. These concepts can be quite confusing at first, and its pays the student to read and review each concept slowly and attentively.

Axons from nerve cells in the eye pass posteriorly in the optic nerve (Appendix III, Figure 6). At the chiasma, those from the nasal retinal field cross over to join the axons from the temporal retinal field, which do not cross (Figure 1). Together they continue posteriorly in the optic tract and end in the lateral geniculate body of the diencephalon. Here they synapse with neurons that sweep out to form the optic radiations, which end in the visual cortex of the occipital lobe. This cortex begins at the occipital pole and is situated on the *cuneate* and *lingual gyri,* which border on the *calcarine fissure* (Figures 1 and 2). Thus, the left visual field of each eye is represented on the right occipital cortex, whereas the right visual fields are represented on the left occipital cortex (Figure 1). The lens also inverts the upper visual field onto the lower part of the retina and vice versa (Figure 2). This pattern is maintained throughout the pathway, so that the cuneate gyrus, which is above the calcarine fissure, receives impulses from the lower visual field, and the lingual gyrus, which is below the fissure, gets impulses from the upper visual field (Figure 2). Finally, the *macula*—the central area of the retina where vision is the sharpest—sends its impulses to the occipital poles (Figure 1).

CLINICAL ASPECTS

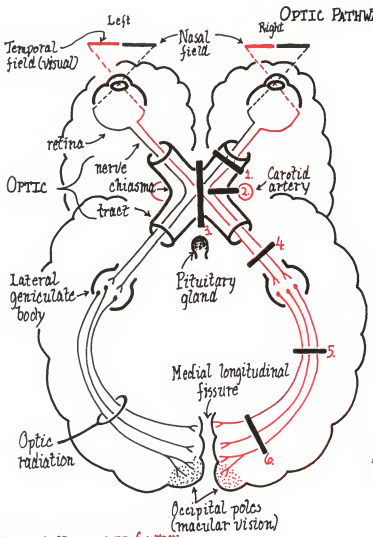
In an eye examination, the fields of vision of each eye are tested and mapped out. If, for example, the right optic nerve is damaged (Example 1, Figure 1) both fields of vision of that eye are affected—in short, *anopsia* or *blindness* of the right eye results.

Example 2 in Figure 1 illustrates how an *aneurysm* of the right internal carotid artery, which lies adjacent to the lateral part of the optic chiasma, can interfere with the temporal axons from the right retina, thus producing *hemianopsia* (half-blindness) in the right eye. Because the visual field affected is the nasal field, we speak of a nasal hemianopsia of the right eye, or right nasal hemianopsia.

Example 3 in Figure 1 shows how the pituitary gland, lying below the optic tracts near the chiasma, can develop an expanding tumor that presses on the decussating nasal axons. This can produce hemianopsia in the temporal visual field of both eyes—a *bitemporal hemianopsia.*

Examples 4, 5, and 6 in Figure 1 illustrate how a lesion in the right optic tract, the right optic radiations, or the right visual cortex can produce loss of vision in the left visual fields of both eyes, which is called a *left homonymous hemianopsia.*

Because the visual field of each eye is divided into nasal and temporal parts plus upper and lower parts, the term 'quadrant' is often used to denote them (e.g., upper quadrant or lower right quandrant). There can also be various quadrantic anopsias.



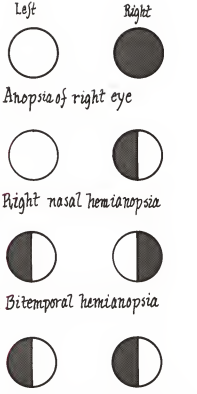


Figure 1. Horizontal Section of Brain

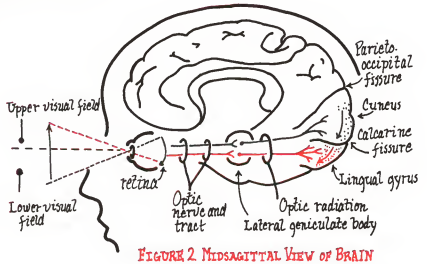


Figure 2. Midsagittal View of Brain

Plate 15-1.

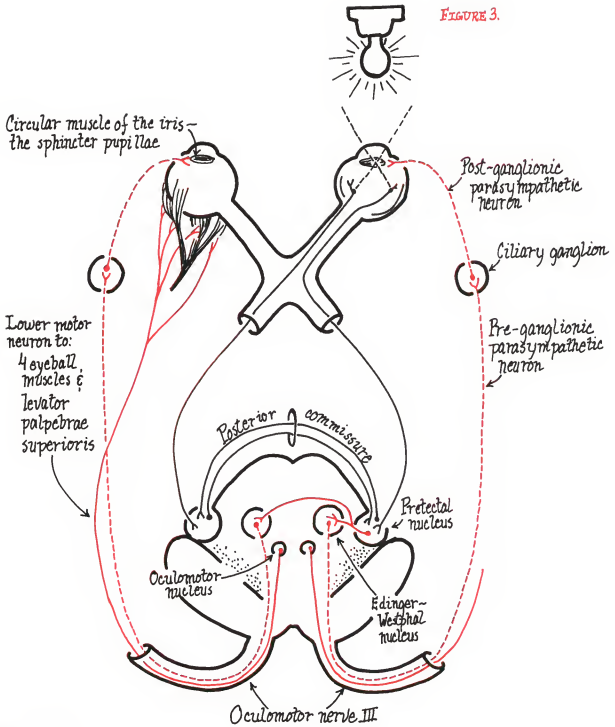


FIGURE 3.

PLATE 15-2.

OPTIC REFLEXES

If light from a small source, such as a pencil flashlight, is shone into one eye from a short distance, there will be pupillary reflex constriction in both eyes—a reaction known as a *consensual reflex.* As you have just learned, optic tract fibers end in the lateral geniculate body. However, about 1% of them peel off just before reaching the geniculate body and terminate in the pretectal nucleus of the midbrain (Figure 3). From here internuncial fibers pass to the parasympathetic Edinger-Westphal nucleus, which then automatically discharges motor stimuli to the circular muscle of the iris, causing pupillary constriction (Figure 3). Therefore, there are three ways that light impulses from one eye can cause reflex constriction in both pupils:

1. Some optic fibers from the right eye's nasal retinal field cross in the chiasma to reach the contralateral pretectal nucleus (Figure 3).
2. The right and left pretectal nuclei are interconnected by commissural neurons that pass through the posterior commissure. Therefore, a stimulus reaching one nucleus is relayed to the other (Figure 3).
3. Each pretectal nucleus sends out fibers to both the right and left Edinger-Westphal nucleus (Figure 3).

CLINICAL ASPECTS

The pupillary light reflex is one of the most useful and important reflexes in medical practice. It occurs even when a person is unconscious. If it cannot be elicited, a serious condition in the CNS, especially the brainstem, is indicated.

In a person who is dying the pupils often dilate markedly and do not contract to light. On the other hand, in persons who have taken narcotics such as heroin or morphine the pupils constrict greatly ("pinpoint pupils") and do not dilate. This fact is often used by police officers and physicians to determine whether a person is intoxicated on such drugs.

Pupillary reflexes—dilation (mydriasis) and/or constriction (myosis)—are also very important in general anesthesia. Here, according to the degree of dilation or constriction (as well as other signs), the anesthetist knows very accurately what stages and planes the patient is in. (Stages and planes refers to the degrees of depth of unconsciousness.)

The Edinger-Westphal nucleus is also concerned with the accommodation reflex, whereby the lens of the eye accommodates itself for near and far vision. From this nucleus, motor stimuli are sent out over the pre- and postganglionic fibers to reach the ciliary muscle that controls the anteroposterior (front-to-back) diameter of the lens. This is a complex reflex that involves cortical areas as well as the *nucleus of Perla,* which is concerned with the convergence of the eyes.

Chapter 16

THE OLFACTORY SYSTEM

Many lower animal forms, such as dogs, deer, amphibians, and certain birds, depend primarily on the sense of smell to locate food, to distinguish friend from foe, and to attract the opposite sex. Consequently, the olfactory system is very highly developed in these animals and is closely connected to the aggressive drive, because this drive is necessary to obtain the above-mentioned objects. In humans, the sense of smell is probably the least important of the major senses, but its pathways, carried over from lower forms, are the most complex of the nervous system. Furthermore, there is a great deal of contradictory data, based on experiments in animals, and the beginning student who opens most neuroanatomy texts finds himself immersed in a welter of conflicting theories couched in the most obtuse and strange terminology. (It is a rule of thumb in science, medicine, and other subjects that the more theories and the more terminology there are concerning a subject, the less is known about it; psychology, psychoanalysis, and economics are excellent examples of this.) This chapter discusses the basic, generally agreed-upon facts concerning the olfactory system, and touches very lightly on the experimental data.

In the epithelial tissue of the nasal cavity are located receptor cells sensitive to smell. These first-order neurons, which are bipolar, pass up into the olfactory bulb, where they synapse with second-order neurons whose axons form the olfactory tract. This tract runs posteriorly and then bifurcates into lateral and medial olfactory tracts, or *stria* (Figures 1 and 2). The area between the bifurcating stria forms the anterior perforated area (Figure 2). The axons of the medial olfactory stria terminate in the paraolfactory (septal) area and the anterior perforated area, while some enter the anterior commissure and cross over to terminate in the contralateral septal area (Figures 1 and 2). The fibers of the lateral olfactory stria end in the cortex of the uncus and in the underlying amygdaloid nucleus (Figures 1 and 2). It is believed that the septal area, the anterior perforated substance, and the cortex of the uncus are the cerebral areas concerned with the "interpretation" of smell (i.e., the primary olfactory areas or centers).

In humans the sense of smell can trigger various emotions and their related reflexes. For example, the smell of good food causes pleasure and salivation, whereas that of rotten eggs causes disgust, nausea, and even vomiting. An enticing perfume may result in sexual arousal (isn't that its basic purpose?), whereas other odors may elicit long-forgotten memories. The major reflex pathways are as follows: From the amygdaloid nucleus, fibers collect in a bundle, the *stria terminalis*, which loops around and terminates in the hypothalamus (Figure 3). The amygdala also sends short fibers to the adjacent hippocampus, where they synapse with neurons that form a large bundle, the *fornix*. This distinctive tract curves up and around to end in the mammillary bodies of the hypothalamus (Figure 3). Finally, from the septal or paraolfactory area, short fibers pass to terminate also in the hypothalamus (Figure 3). It isn't surprising that all these reflex pathways end in the hypothalamus, for, as shown in the following chapter, this is the main coordination and reflex-discharge center for many sensations such as smell, taste, and emotions, as well as control center of the autonomic nervous system. Reflex-discharge pathways carry smell sensations from the hypothalamus to the appropriate motor nuclei and reticular areas in the brainstem. The two main tracts are the mamillotegmental and the dorsal longitudinal fasciculus (Figure 3). Finally, from the mammillary bodies there is a large bundle, the mamillothalamic tract, which ends in the anterior group of the thalamic nuclei. From here the impulses are

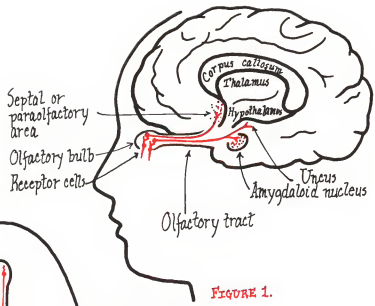


FIGURE 1.

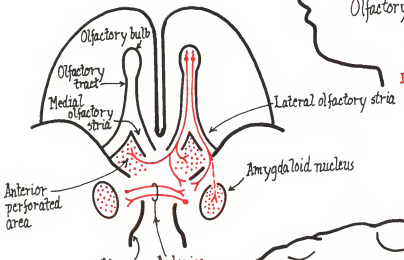


FIGURE 2.

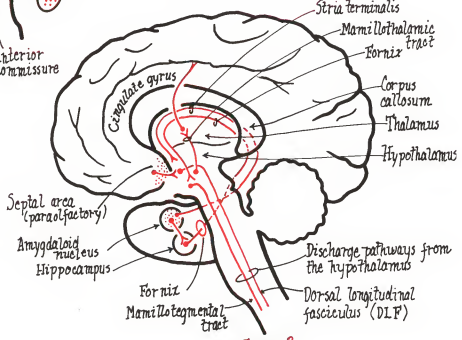


FIGURE 3.

PLATE 16-1

relayed to the cingulate gyrus (Figure 3). In spite of much experimental work, no functional significance of this pathway has been discovered.

CLINICAL ASPECTS

Loss of smell results from damage to the receptor cells, the olfactory bulb, or the olfactory tract, and is known medically as *anosmia.* Lesions of the temporal lobe in the area of the uncus and the amygdala often produce olfactory hallucinations, epileptic seizures, or a combination of both, known as *uncinate fits* when the epileptic fit is preceded by an olfactory aura that has an unpleasant smell.

In monkeys the removal of the amygdala results in very docile, somnolent animals, whereas in cats the same procedure produces animals who are very aggressive in unprovoked situations—a condition known as sham rage. However, in both species the procedure causes a greatly increased sexual drive.

DIAGNOSTIC TESTS

The sense of smell is tested separately in each nostril. Close one nostril and then pass, successively, vials containing various nonirritative substances such as pine oil, coffee, and perfume under the open nostril. Ask the patient if he or she smells the substance and can identify it. A gradual unilateral loss of smell may indicate the presence of a tumor in the frontal lobe.

A good sense of smell is also a useful diagnostic "tool" for the physician or paramedic. For example, the underlying cause of a patient's coma may be deduced by the odor of the breath. In diabetic coma the breath smells like sweet-spoiled fruit. Alcohol has its own well known smell (vodka being excepted), whereas uremic coma produces a uriniferous odor. In hepatic coma there is a stinking musty smell to the breath.

ROBERT D. HENDRICKS

Chapter 17

THE RETICULAR SYSTEM

The reticular system is a phylogenetically old system that is divided anatomically and physiologically into two parts—a descending and an ascending formation.

DESCENDING RETICULAR FORMATION

The descending reticular formation is a system concerned with:

1. Relaying impulses from the hypothalamus to various target organs of the autonomic nervous system
2. Relaying involuntary motor impulses from the extrapyramidal systems to voluntary muscles.

Scattered deep in the brainstem are the groups of diffuse nuclei or areas (some authors and investigators call them "formations") belonging to this system. In the midbrain they are called the *deep* and *dorsal tegmental nuclei;* in the pons they are called the *central tegmental nucleus;* and in the medulla they are the *central* and *inferior nuclei.* Some books also mention other descending reticular nuclei or call them by different names, but the main point to grasp is not the exact number of nuclei but the fact that they exist and their function.

These nuclei or formations receive stimuli from the hypothalamus fiber tracts, such as the dorsal longitudinal fasciculus and the mamillotegmental tract (Figure 1). In addition, various basal ganglia, such as the globus pallidus, the substantia nigra, and the subthalamic nucleus, project fibers that terminate in these nuclei. Lastly, the vestibular system, which is also extrapyramidal, sends some of its fibers to the reticular nuclei (see Chapters 10 and 11).

These incoming fibers synapse with neurons whose axons then leave the reticular nuclei and form the lateral and medial reticulospinal tracts. These are descending, crossed and uncrossed, multisynaptic pathways that travel down to all levels of the spinal cord in the lateral and ventral white columns. In the cord they synapse either on the ventral horn cells, which form the final common pathway, or on the preganglionic neurons in the intermediate gray horn.

ASCENDING RETICULAR FORMATION

The ascending reticular formation, better known as the reticular activating system, is concerned with degrees of conscious alertness as well as with sleep. Situated in the medulla, pons, and midbrain are groups of poorly defined nuclei connected to each other by a chain of multisynaptic neurons. Because the nuclei and their interconnecting chain have a diffuse and poorly defined appearance, they were given the name *reticular system.*

All the major sensory pathways (e.g., the spinothalamic, for pain, temperature, touch, and pressure; the auditory; the visual) send collateral axons that end in the nuclei of the reticular activating system. These nuclei then send the sensory stimuli they have received up the multisynaptic chain, which ends primarily in a group of nuclei of the thalamus known as the midline group. As has already been shown, the thalamus serves as a relay center for many sensory pathways as well as motor ones, and it is not surprising that it also serves as a relay for the reticular activating system. From the thalamic midline, nuclei impulses are relayed up to the cerebral cortex, where they influence states of mental alertness and sleep. Exactly how these impulses are relayed and what specific regions of the cerebral cortex they reach are not known.

Sleeping animals and humans exhibit a characteristic electroencephalogram (EEG) wave pattern, but if the reticular activating nuclei of sleeping animals are experimentally stimulated, the animals awaken, and we see that the change from sleep to wakefulness is accompanied by a change in the EEG wave pattern. In animals that are al-

FIGURE 1. SCHEMATIC DIAGRAM OF THE DESCENDING RETICULAR FORMATION

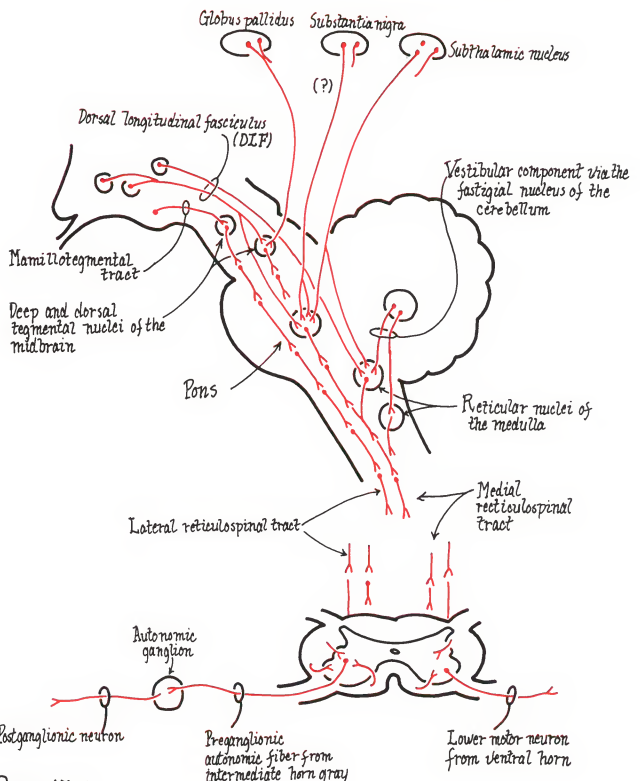


PLATE 17-1.

ready awake, stimulation of the reticular activating nuclei produces states of greater alertness accompanied by characteristic changes in the EEG pattern. It is therefore assumed that alertness and/or sleep is largely dependent on the amount of stimuli reaching the cerebral cortex via the reticular activating system. If the amount of stimuli from the outside world is reduced, there will be a lowering of alertness, and sleep may result. On the other hand, an increase in the amount of stimulation reaching the cerebral cortex via the ascending reticular formation results in greater alertness. In a nutshell, we may say that:

↓ stimulation from various sensory systems into the reticular nuclei → ↓ amount of stimulation to the thalamic midline nuclei → ↓ stimulus to cerebral cortex → ↓ alertness and/or sleep

This simple outline is just one aspect of an extremely complex picture, most of which is still unrevealed to us. One should not conclude that sleep or alertness is totally dependent on the state of the reticular activating system; there are many other factors (e.g., metabolic and psychological factors) that play a part in alertness and sleep. There are also many factors that we know nothing about, the discovery of which will throw new light on the problem of sleep, alertness, and consciousness.

CLINICAL ASPECTS

Although there is no known center for sleep or consciousness, it is believed that the brainstem is primarily involved because damage to it often produces states of unconsciousness or coma. (This is known by post mortem examination of the brain.) The author has a friend who was sitting in a car that had stopped for a red light. It was struck from behind by another car, and the driver suffered a severe whiplash—with no other known injuries—and has been in a coma for over 2 years.

Coma

This is a state of unconsciousness resembling sleep from which the individual cannot be roused even by the strongest stimuli. Coma varies in degree. For example, in deep coma there is a complete absence of response to stimuli and most reflexes are lost. In lighter stages the patient may respond to sounds, some reflexes and movements are present, and the eyes may be open. Many pathologic conditions can produce coma or comatose states, but the three most common causes are alcoholism or other drug intoxications, injuries to the head, and cerebrovascular accidents (strokes).

Stupor

This is a state of unconsciousness from which the patient is aroused with difficulty and when awake is mentally confused.

Concussion

This is usually defined as a transient state of unconsciousness caused by a sudden sharp blow to the head. Upon awakening there may be episodes of vomiting.

Chapter 18

THE HYPOTHALAMUS

The hypothalamus is one of the smallest areas of the brain, yet no other region has so many different and vital functions. As its name indicates, it lies beneath the thalamus. It is seen well in a midsagittal section (Plates III and IV in Appendix II), where it extends from the lamina terminalis to the midbrain. Separating it from the overlying thalamus is a shallow groove, the *hypothalamic sulcus.* The hypothalamus thus forms the lateral wall of the lower part of the third ventricle and is also seen in cross-sections (Plate VII in Appendix II). If one looks at the base of the brain, the hypothalamus is seen as forming the area that lies posterior to the optic chiasma and includes the infundibulum and mammillary bodies. Packed into this small region are many nuclei and areas (Figure 1) that are concerned with such functions as temperature control, sleep, water metabolism, secretion of hormones, control of blood pressure, hunger, and maintenance of balance between the sympathetic and parasympathetic divisions. It also plays a part in emotional reactions and possibly other situations.

HEAT REGULATION

The anterior hypothalamic area is concerned with heat regulation of the body. When there is an increase in body temperature, the heated blood passes through the anterior hypothalamic area and sets off a mechanism that facilitates heat loss (Figure 2). Fibers leave the anterior hypothalamic area and join the dorsal longitudinal fasciculus (DLF), which is the major descending pathway from the hypothalamus. The DLF terminates in the descending reticular nuclei of the brainstem, where it synapses with neurons of the medial and lateral reticulospinal tracts. These then descend the cord and stimulate the sympathetic nervous system and voluntary muscles. Other fibers of the DLF terminate in, and stimulate, the cardiac and respiratory centers in the medulla. The results of all these stimuli are the following reactions, which serve to reduce body temperature:

1. Dilation of peripheral blood vessels beneath the skin, with a subsequent increase in heat radiation.
2. An increase in sweating, which reduces heat. (Evaporation is a cooling process.)
3. Increase in respiratory rate, with "blowing off" of hot air from the lungs.
4. A decrease in the body's metabolic rate.
5. Increases in peripheral blood flow accompanied by increased heat dissipation.

If we experimentally destroy an animal's anterior hypothalamic region, it becomes unable to respond to heat increases in its environment. Thus, when the temperature rises, its body temperature rises, and eventually it will die from heat prostration.

Cold regulation is controlled by the posterior hypothalamic area. When the temperature of the environment drops, the body becomes cooler. The cooled blood passes through the posterior hypothalamic area (Figure 2) and sets off a mechanism that is directly the opposite of the one just discussed. The pathways are basically the same—the DLF, reticular nuclei, and reticulospinal tracts. The reactions set off to conserve body heat are as follows:

1. Peripheral vasoconstriction, with a subsequent decrease in the amount of heat lost by radiation
2. A decrease in peripheral blood flow
3. An increase in body metabolism
4. Shivering of voluntary muscles. Shivering is work in which energy, in the form of heat, is produced. (There is, of course, also energy of motion.)
5. A decrease in the respiratory rate.

Experimental lesions in the posterior hypothalamic area produce animals that are unable to adjust to cold environments, and their bodies become as cold as the surroundings.

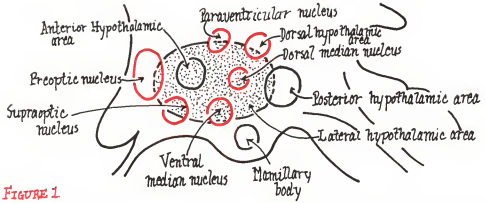


FIGURE 1

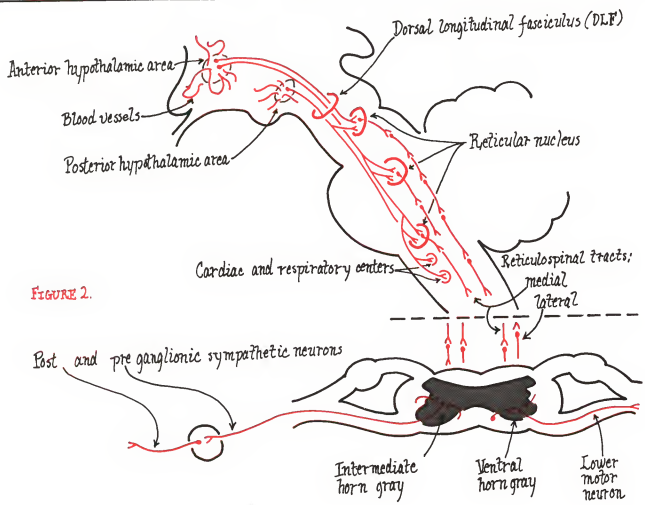


FIGURE 2.

PLATE 18-1.

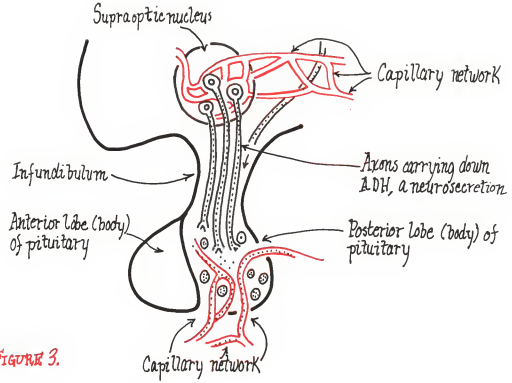


FIGURE 3.

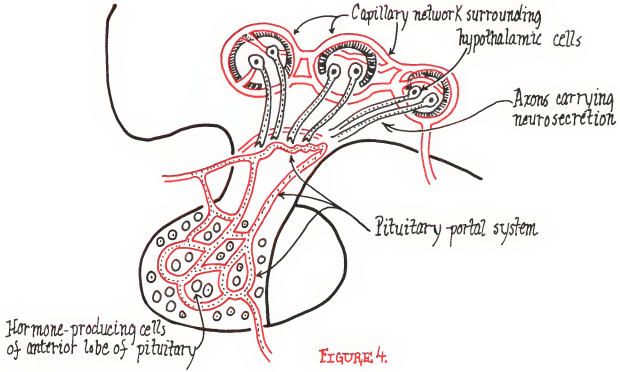


FIGURE 4.

PLATE 18-2.

WATER BALANCE

The hypothalamic mechanism for maintaining water balance is one of the most interesting regulatory mechanisms of the body. It is known that a hormone from the posterior pituitary body, called antidiuretic hormone, or ADH, acts on the distal convoluted tubules of the kidney, causing resorption of water. If the amount of ADH produced is reduced, a pathologic condition known as diabetes insipidus results. In this disease the patient will urinate 18–20 liters of urine per day in stead of the normal 1–2 liters. The regulatory mechanism in the production and release of ADH is a function of the supraoptic nucleus of the hypothalamus. The cells of this nucleus and possibly those of the paraventricular nucleus produce ADH, and this neurosecretion passes down the axons of the neurons, via the infundibulum, to reach the cells of the posterior pituitary (Figure 3), where the ADH is either stored or released into the capillary network.

If there is a reduction in the amount of water in the blood, the cells of the supraoptic nucleus, which are sensitive to such a change, will produce and release more ADH. This results in more water being resorbed by the kidney tubules and its conservation by the body. On the other hand, if there is a state of hydration, the cells of the supraoptic nucleus react by decreasing the production and release of ADH. This decrease in the production and release of ADH results in a decrease in the amount of water resorption by the kidneys, and therefore a greater amount is urinated.

INFLUENCE OF THE HYPOTHALAMUS ON THE SECRETION OF HORMONES FROM THE ANTERIOR LOBE OF THE PITUITARY

There is much evidence that cells of the hypothalamus *can in part* influence the secretion of various hormones of the anterior lobe of the pituitary gland. The mechanism resembles that of water metabolism. Neurosecretory cells of the hypothalamus are very sensitive to the blood concentration of the various anterior lobe hormones. In response to a decrease, these neurons produce a neurosecretion that passes down the axons. However, the axons terminate in the region of the infundibulum, and here the neurosecretion is "picked up" by a pituitary portal system (Figure 4). This carries the neurosecretion to the anterior lobe, where it stimulates the cells to produce the various hormones. One must not conclude that the hypothalamus is the only, or principal, regulator of hormones from the anterior lobe. There are other mechanisms, such as a direct feedback control, as well as mechanisms that are not yet clearly understood.

HYPOTHALAMIC DISCHARGE IN EMOTIONAL STATES

Various emotional states, such as anger or fear, result in physiologic reactions. The hypothalamus is a center for the control and discharge of such reactions. For example, when one sees or hears something that evokes an angry reaction, the stimuli first reach various areas of the cerebral cortex, such as the visual or auditory centers, the memory centers, or the personality area of the frontal lobe, all of which are interconnected by association tracts. From the cerebral cortex, especiallly its frontal lobe, there is a discharge pathway to the hypothalamus. From the latter, the major descending pathway is the dorsal longitudinal fasciculus, which arises from all the hypothalamic nuclei, and areas except the supraoptic and ventral median nucleus. (A minor pathway is the mamillotegmental tract.) The dorsal longitudinal fasciculus leaves the hypothalamus and passes down the length of the brainstem, where it gives off branches to all the descending reticular nuclei; all the parasympathetic nuclei of cranial nerves III, VII, IX, and X; the respiratory and cardiac centers; and the motor nuclei of the cranial nerves (Figure 2). From the reticular nuclei emerge the lateral and medial reticulospinal tracts, which descend the cord to supply the autonomic nervous system as well as the voluntary muscles. Thus, we see the complex interrelationship that exists between various parts of the brain, and one must proceed with great caution in applying new surgical or other techniques, such as lobotomies.

The hypothalamus is also involved in the olfactory reflex system (see Chapter 16). Finally, experimental work in animals has demonstrated that destruction of the ventral median nucleus produces animals with voracious, almost insatiable, appetites, whereas destruction of the lateral hypothalamic area produces animals that have no appetite. Clinically, we see that some patients who have tumors of the hypothalamus lose their appetites and become emaciated.

CLINICAL ASPECTS

Many weight-reducing compounds contain substances that inhibit the appetite centers. This action results in decreased appetite—and, hopefully, reduced weight.

Chapter 19

THE CEREBRAL CORTEX

The cerebral cortex is most highly developed in humans. It is responsible for the qualities that distinguish humans from other animals—for example, the ability to use the hand for skilled and intricate movements, a very high level of speech, symbolic thought, personality, and conscience. We know all this because if certain areas of the cortex are damaged these qualities are lost or greatly reduced.

In submammalian species the cerebral cortex is small and concerned almost exclusively with smell, which, as has already been described, is for them one of the most important sensations. The thalamus is the main sensory receptor area, and the basal ganglia and subthalamic nuclei serve as the motor discharge areas. Because fine, complicated voluntary movements are not seen in these lower forms, the cerebellum is primarily a center for equilibrium, which is a function of its flocculonodular lobe. As one ascends the evolutionary ladder, the cerebral cortex enlarges and takes on other functions. For example, the main sensory area is now localized in the postcentral gyrus and its former center, the thalamus, now becomes a center that relays the sensory impulses from the body to the cortex. With the appearance of the cerebral motor cortex, the basal ganglia in humans become areas of crude motor activity. Parallel with the development of this motor cortex there is a great development of the cerebellum as a coordination center for muscle activity, but the floccular nodulus remains the center for body equilibrium. In humans some functions, such as smell, decrease greatly in importance, although the complicated pathways remain—and drive medical students up the walls.

With the increase in complexity and functions there is an increase in the number of neurons, and the area of cerebral cortex increases to such a degree that the cortex, in order to expand in the same volume area, is thrown into folds, giving the characteristic appearance of gyri and sulci (see Appendix III, Figures 1–5). (In lower forms, such as the rat, the surface of the cerebral cortex is smooth.) This same principle is used by restaurant owners in high rent areas—instead of having straight counters they make them convoluted, and thus squeeze in more customers.

As has been mentioned throughout this text, certain areas of the cortex have specific functions. The precentral gyrus (area 4) is concerned with initiating voluntary movements, whereas the postcentral gyrus (area 3, 1, 2) is the primary somatic sensory reception center. The occipital pole and the area on both sides of the calcarine fissure (area 17) form the primary visual receptor center. Areas 41 and 42, situated on the superior temporal gyrus, are the primary auditory reception center. Damage to any of these areas results in a loss of function such as paralysis, anesthesia, or blindness. In addition, area 8, lying anterior to area 6 in the frontal lobe (Figure 1), is concerned with voluntary conjugate movements of the eyes. The frontal poles and the areas surrounding them are the site of personality. A person who suffers injury to this area (say, following a car accident) will probably undergo personality changes. The author remembers a case of a very friendly and pleasant social worker who suddenly and for no apparent reason became very argumentative and abusive, until her death a short while later. Autopsy revealed an expanding tumor in the frontal lobe that had caused both her death and the marked changes in character.

In the mid-1930s a Portuguese neurosurgeon, Moniz, introduced the procedure of cutting or removing parts of the frontal lobes—a lobotomy—as a means of treating severely psychotic patients. With hardly a murmur of dissent, this procedure was widely hailed (Moniz received the Nobel prize for it in 1949) and widely practiced. True, after the operation many of the patients were quieter and more docile, but they also lost all initiative, became indifferent to their surround-

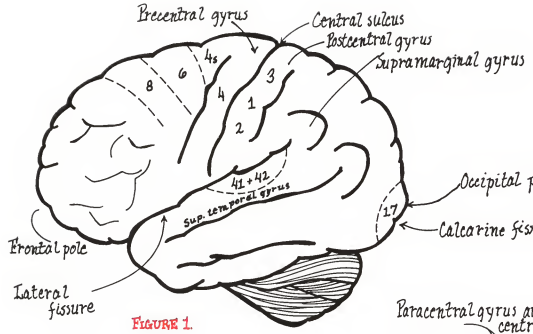


FIGURE 1.

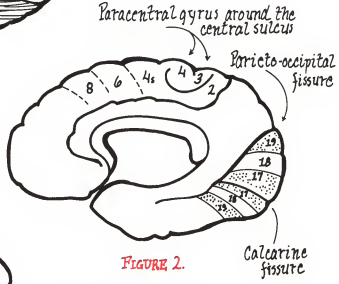


FIGURE 2.

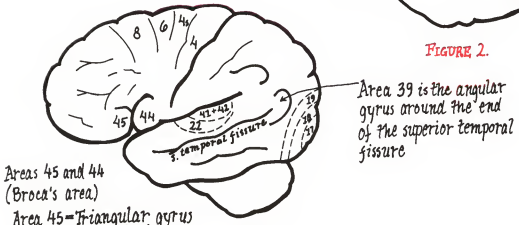


FIGURE 3.

PLATE 19-1.

ings, defecated and urinated in public, and showed other behavioral disturbances. Today this barbarous operation is thoroughly discredited. Any surgeon contemplating it should take into account, among other things, the fact that Moniz was almost murdered by a former patient who was distraught over his new state. This was one case where the operation was a success but the surgeon nearly died!

Surrounding each of the primary cortical areas and closely allied with them are *associated areas.* Around the visual area (area 17) there are areas 18 and 19, which have several functions. First, they are concerned with "interpreting" the visual impulses that reach area 17. We see round, red objects in front of us, and areas 18 and 19 interpret them as apples. This interpretation is called *gnosis,* from the Greek word meaning "to know." Area 19 is concerned with automatic following movements of the eyes, which occur when an object, such as a jet, suddenly comes into the visual field and the eyes "lock in" and follow it. Associated with area 4 is area 4s, the suppressor band, and area 6, which helps with voluntary movements. Area 22 is the auditory association area. If this area is damaged on the dominant side (the left hemisphere in most people, including those who are left-handed), the result is the condition known as *word deafness,* or *auditory aphasia,* which is discussed in the following section.

SENSORY (RECEPTOR) APHASIA

Aphasia is defined as the inability to understand or express the symbols connected with language; there are two basic kinds: sensory and motor. If one traces the superior temporal sulcus to its posterior end, the gray matter surrounding this end is the *angular gyrus* (area 39) of the parietal lobe (Figure 2). Damage to this area on the dominant cerebral hemisphere produces a condition known as *visual aphasia,* "word blindness," or *alexia.* In this condition the patient sees the printed words but cannot read them—they are meaningless lines. This condition is equivalent to a Westerner looking at Chinese writing; all the Westerner sees are curved lines and characters that have no specific meaning.

Area 22, surrounding the primary auditory reception areas (41 and 42), is the auditory reception area. If it is damaged, again on the dominant cerebral hemisphere, *auditory aphasia,* results. A patient with this condition experiences sound without any meaning—the same thing as when you hear speech in a completely foreign language. The patient can hear you speaking but cannot understand what is being said. *Wernicke's aphasia* is a condition of both auditory and visual aphasia.

MOTOR APHASIA

On the inferior frontal gyrus, in the triangular and opercular regions, are found areas 44 and 45, also known as *Broca's area* (Figure 3). If these areas are injured in the dominant hemisphere in an adult, they produce a condition in which the patient is unable to talk, even though the vocal muscles are not paralyzed. The patient knows what he or she wants to say, but all that comes out is garbled sound or one word repeated over and over again. One might speculate that the memory engrams connected with speech have been destroyed. If the damage occurs in childhood, the child can be taught to speak by utilizing the nondominant cerebral hemisphere.

APRAXIA

Apraxia is the inability to carry out purposeful, learned, voluntary acts although there is no paralysis present. It also involves the association areas. When told to take out his or her keys and open the door, the patient might pull out a coin or comb and try to put it into the keyhole. If the damage involves the loss of writing ability, it is known as *agraphia.*

AGNOSIA

Agnosia is the inability to recognize things even though one sees them. For example, a patient can walk down the street, see some broken glass in the way, and walk around it. However, when you ask what it is he walked around, the patient doesn't know.

These conditions may sound strange, but many things concerning the cerebral cortex are so. As can readily be appreciated, the subject of aphasias and apraxias, as well as other cerebral conditions such as epilepsy, are not as simple as has been presented here, but are very complex matters that have psychological aspects as well. Our "hard fact" knowledge is very restricted, and because experiments can't be performed easily on the human cortex, what little information we have comes from pathologic cases and autopsies. The reader who wishes to learn more about the telencephalon should consult *Correlative Anatomy of the Nervous System,* by Crosby, Humphrey, and Luer, which has nearly 200 pages on the subject and over 1,300 references. There are also books available that are devoted to individual subject matters, such as epilepsy and EEG.

Chapter 20

THE MENINGES

Brain tissue, having the consistency of a heavy pudding or custard, is the most delicate of all body tissues. For protection, this vital organ is located in a sealed bony chamber, the *skull**. To protect it further from the rough bone and from blows and shocks to the head, the brain is enveloped by three membranes, called the *meninges.* The outermost covering is the tough, thick *dura mater,* which is adherent to the inner surface of the bone (Figure 1). In fact, it forms the periosteal layer of the calvarium. Beneath the dura mater is the middle covering, the thin and filamentous *arachnoid.* The third and innermost layer is the very thin, delicate, and capillary-rich *pia mater,* which is attached directly to the brain and dips down into the sulci and fissures (Figure 1).

Although the dura matter is closely applied to the inner bone surface, it can in certain instances separate from it, creating an area between the two known as the *epidural space* (see "Clinical Aspects" at the end of Chapter 21). Between the dura mater and the underlying arachnoid is a very narrow subdural space filled with a small amount of serous fluid that acts as a lubricant, preventing adhesion between the two membranes (Figure 1). Separating the arachnoid from the pia mater is a relatively large gap, the *subarachnoid space* which is filled with cerebrospinal fluid (CSF; Figure 1). This clear, lymph-like fluid fills the entire subarachnoid space and thus surrounds the brain with a protective cushion that absorbs shock waves to the head. As a further means of protection there are fibrous filaments known as the *arachnoid trabeculations,* which extend from the arachnoid to the pia and help "anchor" the brain to prevent it from excess movement in cases of sudden acceleration or deceleration (Figure 1). In the fluid-filled subarachnoid space are situated the cerebral arteries and veins (Figure 1). The pia mater is so closely attached to the underlying brain that there is no space, potential or otherwise, between the two. In this manner the pia mater acts as a restraining agent that holds the brain tissue together and prevents it from separating.

The dura mater dips down into the median longitudinal fissure, and this dural fold, lying between the cerebral hemispheres, is called the *falx cerebri* (Figures 1 and 2; see also Appendix III, Figures 2–5). The dura also dips into the space between the cerebellum and the overlying occipital lobes. This is called the *tentorium cerebelli* (Figures 1 and 2) because it forms a tentlike covering over the cerebellum. Finally, the dura mater dips between the two cerebellar hemispheres to form the *falx cerebelli* (Figure 2). All the meninges, the subarachnoid space, and the CSF also extend down and surround the spinal cord.

CLINICAL ASPECTS

Meningitis is an infection of the meninges; usually it is the arachnoid and pia mater that are attacked (leptomeningitis). As you probably know from personal experience, an infected and inflamed area is very sensitive, and any pressure or stretching of it causes great pain. In meningitis, when there is an attempt to flex (bend) the neck and thereby stretch the meninges, the muscles of the neck contract strongly to prevent the bending and subsequent pain. This phenomenon of muscle contraction to prevent stretching of inflamed structures is known as *guarding.* In cases of suspected meningitis, the physician tries to bend the neck of the supine patient. If the neck cannot be bent or if bending is accompanied by pain, this is a key sign that meningitis is probably present.

*The English word "skull" is related to the Scandinavian word *skøl,* now a popular drinking toast. The Vikings used to cut off the top part of their victim's skull and use it as a drinking cup.

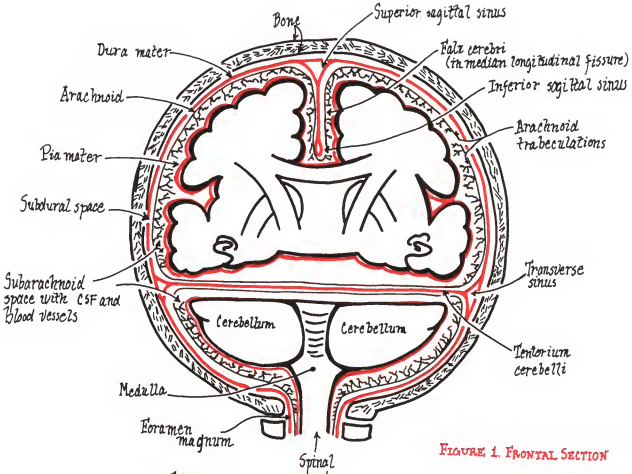


FIGURE 1. FRONTAL SECTION

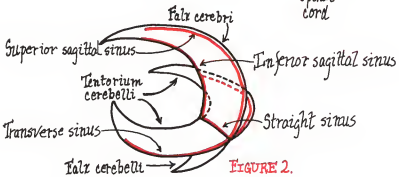


FIGURE 2.

PLATE 20-1.

Chapter 21

BLOOD SUPPLY TO THE BRAIN

As mentioned in Chapter 1, nerve cells do not regenerate. They also need a constant, adequate supply of blood, and any interruption of it or injury to the vascular tree can quickly lead to irreparable, lifelong damage or death. Because such injuries are commonly encountered in medical practice, knowledge and understanding of CNS vascularity is essential. Two pairs of arteries, the vertebrals and the internal carotids, are the only suppliers to the brain. The *vertebral arteries* enter the skull through the foramen magnum and pass along the ventral surface of the medulla (Figure 1). After giving off the *anterior* and *posterior spinal arteries* as well as the *posterior inferior cerebellar artery,* they join together to form the *basilar artery,* which passes up to the beginning of the pons, where it bifurcates into the *posterior cerebral arteries.* These sweep back to supply the posterior part of the cerebral hemispheres, especially the medial and basilar surfaces (Figures 1, 2, and 3). In its course the basilar artery gives off the *anterior inferior cerebellar artery, pontine branches,* and the *superior cerebellar artery.*

The internal carotid arteries enter the skull through the foramen lacerum and lie adjacent to the lateral border of the opic chiasma (Figure 1). Here they bifurcate into the *anterior* and *middle cerebral arteries.* The anterior cerebral arteries pass forward into the medial longitudinal fissure and then sweep back to the parieto-occipital fissure, thus supplying the medial surface of the hemisphere (Figures 1–3). The *middle cerebral arteries* pass laterally between the temporal and frontal lobes. They emerge at the lateral fissure and fan out to supply most of the lateral surface of the hemisphere (Figures 1 and 2). In their course between the temporal and frontal lobes, the middle cerebral arteries give off the very important *striate arteries,* which help supply the internal capsule with its descending motor tracts (Figure 1). Because the striate arteries are the frequent site of cerebrovascular accidents (CVA), they are known as the "arteries of stroke."

The anterior cerebral arteries are connected to each other by the *anterior communicating artery.* There is also a posterior artery that links the middle cerebral artery with the posterior cerebral artery (Figure 1). Thus, at the base of the brain an anastomotic ring is formed between the vertebral and internal carotid arteries. This ring is called the *circle of Willis.* This formation is important clinically because if one of the arteries becomes occluded, the blood can pass around to reach the deprived area. In addition, the circle of Willis is a frequent site for *aneurysms.* An aneurysm forms when blood pressure at a weakening in the wall causes the artery to balloon out. An aneurysm can press on adjacent structures, such as the optic chiasma, causing visual disturbances (Figure 1, and also refer to Chapter 15).

VENOUS DRAINAGE

Venous blood takes a roundabout circuit in its drainage to the neck. Most of the veins reach the surface of the brain and join larger veins. These cross the subarachnoid space and empty into large venous sinuses located within the dura mater. There is a confluence of these sinuses into each other: the superior sagittal and straight sinuses flow into the transverse, which continues into the sigmoid, which drains into the internal jugular vein of the neck. The superficial middle cerebral vein flows into the cavernous sinus located at the base of the brain (see Appendix III, Figure 5).

CLINICAL ASPECTS

If an artery becomes occluded by an embolism or through vasospasm, the area distal to the occlusion is deprived of its blood supply and the cells quickly die. This usually results in a stroke, the severity of which depends on the artery stopped and the site of occlusion, as well as other factors. Stroke can also occur if an artery ruptures, and, if the hemorrhage is massive, death can occur very quickly.

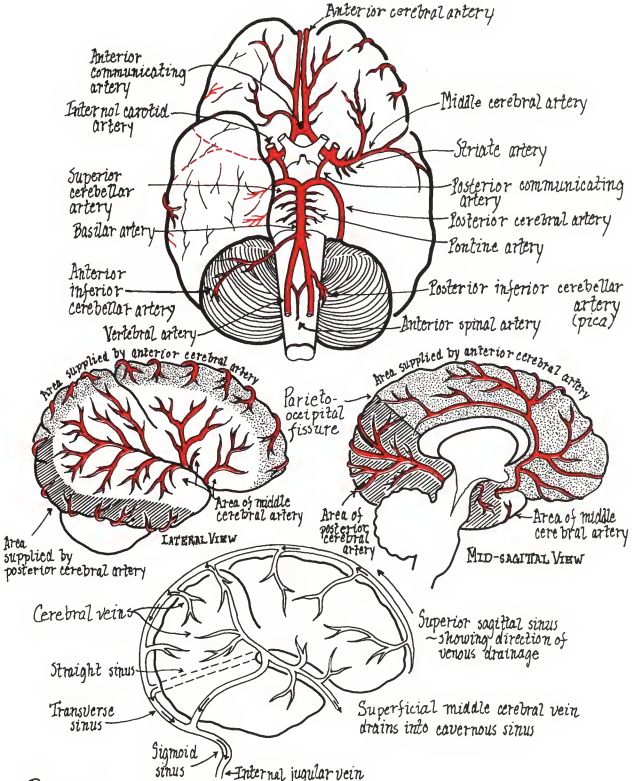

Anterior cerebral artery
Anterior communicating artery
Internal carotid artery
Middle cerebral artery
Striate artery
Superior cerebellar artery
Posterior communicating artery
Posterior cerebral artery
Basilar artery
Pontine artery
Anterior inferior cerebellar artery
Posterior inferior cerebellar artery (pica)
Vertebral artery
Anterior spinal artery
Area supplied by anterior cerebral artery
Parieto-occipital fissure
Area supplied by anterior cerebral artery
Area of middle cerebral artery
Area supplied by posterior cerebral artery
LATERAL VIEW
Area of posterior cerebral artery
Area of middle cerebral artery
MID-SAGITTAL VIEW
Cerebral vein
Superior sagittal sinus ~showing direction of venous drainage
Straight sinus
Transverse sinus
Superficial middle cerebral vein drains into cavernous sinus
Sigmoid sinus
Internal jugular vein

PLATE 21-1.

The middle meningeal artery does not supply the brain, but the dura of the middle cranial fossa. It lies between the dura mater and the skull, and in cases of severe trauma to the head, as in car accidents, jagged bone splinters can cut the artery. Then arterial blood, which is under high pressure, flows out rapidly between the dura and the bone, forming a rapidly expanding pool (an *epidural* or *extradural hematoma*) that presses on the underlying brain (Appendix III, Figure 11). Unconsciousness soon follows, and immediate surgical intervention is needed to prevent death. This condition and others are beautifully illustrated by Dr. Netter in the world-famous Ciba collection of atlases.

Because the veins of elderly people are less resilient and more fragile than in younger individuals, a mild blow to the head can cause a cerebral vein to rupture slightly. Because venous pressure is low, seepage is very slow and the blood usually accumulates between the dura and the arachnoid, forming what is known as a *subdural hematoma.* Weeks later, after the blow has been forgotten about, the slowly expanding hematoma presses on the brain, causing various insidious and nonspecific symptoms such as dizziness, headaches, apathy, falling, confusion, and drowsiness. In the great majority of cases, a CT scan can accurately pinpoint the condition.

Subdural hematomas may also occur in newborns, as great pressure on the head during delivery may rupture a cerebral vein.

Chapter 22

CEREBROSPINAL FLUID AND THE VENTRICULAR SYSTEM

Cerebrospinal fluid (CSF) is a clear fluid filling the entire subarachnoid space. It acts as a protective "liquid cushion" around the brain and spinal cord by absorbing shock waves from blows and falls. In addition, it is a valuable diagnostic aid: by means of a relatively simple procedure known as a spinal tap, the physician can obtain a fresh sample of the fluid, quickly examine it, and get an accurate picture of what is taking place within the skull and brain.

Deep inside the brain is a series of interconnecting chambers—the ventricular system—and it is here that CSF is produced. In each cerebral hemisphere there is a large space, the *lateral ventricle* (see Appendix III, Figures 2–5), which is made up of an *anterior horn*, lying in the frontal lobe; the *body* or main part, lying in the frontal and parietal lobes; a *posterior horn*, in the occipital lobe; and an *inferior horn*, which sweeps down into the temporal lobe (Figures 1 and 2). In each lateral ventricle there is a delicate, lacelike structure, the *choroid plexus* (Figure 3; see also Appendix III, Figure 3), which is composed of pia mater enveloped by the thin membranous ependyma. Due to diffusion and active transport, the CSF passes from the capillary-rich choroid plexus into the ventricular space; it is therefore similar to lymph. The accumulating CSF fills the lateral ventricles and then flows out of them via the interventricular *foramen of Monro* and into the third ventricle (see Appendix III, Figures 3 and 4). This narrow, slitlike space lies in the midline between the walls of the right and left diencephalon (Figures 1 and 2; see also Plates IV and VII in Appendix II). The choroid plexus in the third ventricle also produces CSF, and all of the fluid flows into the narrow aqueduct of Sylvius, located in the midbrain (Figures 1 and 2; see also Plates IV, X, and XI in Appendix II). The aqueduct then empties into the fourth ventricle, in the pons and medulla (Figures 1, 2, and 3; see also Plate IV in Appendix II and Figure 4 in Appendix III). Here also is the choroid plexus, which produces CSF. In the thin roof of the fourth ventricle are three openings—the medial *foramen of Magendie* and the two lateral *foramina of Luschka**. It is through these openings that the CSF leaves the ventricular system and flows into and completely fills the subarachnoid space around the brain and cord (Figures 2 and 3). In certain regions the arachnoid is situated far from the pia mater, and the enlarged subarachnoid space forms areas known as *cisterns*, for example the cisterna magna (Figure 3).

An important question is, if CSF is constantly being produced, what happens to the excess fluid? In the area of the superior sagittal sinus, the arachnoid projects through small openings in the dura mater into the sinus. The accumulating CSF creates a pressure that forces the excess fluid out of the arachnoid projections and into the dural venous blood, which carries it away (Figures 3 and 4). In gross preparations these fine arachnoid projections resemble granules of sugar or salt, and are therefore called *arachnoid granulations*.

CLINICAL ASPECTS

Hydrocephalus

Most often in newborn infants, a blockage may form somewhere in the ventricular system. Consequently, the CSF is unable to flow out and instead accumulates in the ventricles, where it presses on the nervous tissue, causing a thinning out of the brain with a widening of the ventricles (see Appendix III, Figure 8). Because the cranial bones of the

*Magendie is *m*edian; *L*uschka is *l*ateral.

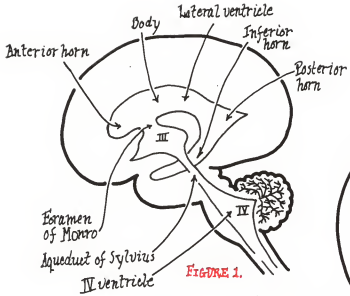


FIGURE 1.

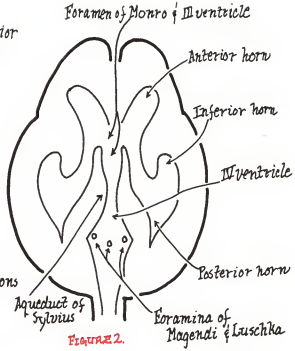


FIGURE 2.

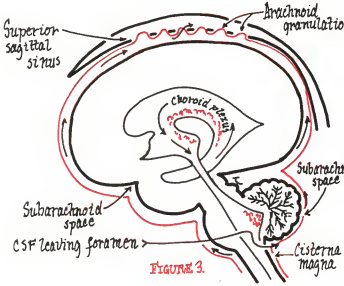


FIGURE 3.

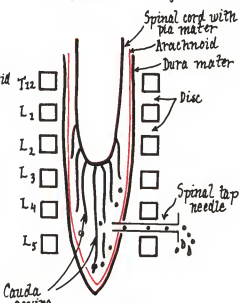


FIGURE 5.

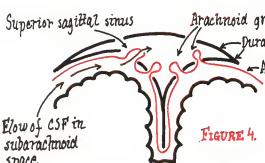


FIGURE 4.

PLATE 22-1.

baby have not yet fused, the expanding, fluid-filled brain separates the bones and the head enlarges tremendously. The exact cause of hydrocephalus is unknown, but it may be due to failure of the foramina or aqueduct to develop, or they may become blocked by a tumor or following *encephalitis* (infection of the brain). Therefore, examination of infants must include a measurement of the circumference of the head, and if it exceeds normal limits then diagnostic tests should be done.

Today excellent therapeutic results are obtained in the treatment of hydrocephalus by neurosurgically implanting a tube (catheter) from the anterior horn of the lateral ventricle to the pleural (lung) cavity. The excess CSF is thereby shunted off and absorbed in the pleural cavity.

Spinal Tap

Because the spinal cord is shorter than the vertebral column, it generally ends at the level of the first or second lumbar vertebra (Figure 5). Therefore, the subarachnoid space below this level can be tapped with no danger of injuring the cord. After giving a local anesthetic, the doctor inserts a sterile hollow needle in between the third and fourth or fourth and fifth lumbar vertebrae, punctures the dura mater, and enters the subarachnoid space filled with CSF. The hollow needle has a plunger, which is pulled out so that the CSF can then drip out. The physician measures the pressure of the intracranial CSF, which normally can reach 200 mm of water. In certain brain diseases the CSF pressure is greatly elevated. However, one must *NEVER* attempt to reduce the pressure by letting out the CSF through a spinal tap, because the sudden downward flow of the released fluid pulls the brainstem into the foramen magnum, causing the almost instantaneous death of the patient.

The spinal fluid obtained by spinal tap is examined for the presence of pus, blood, and bacteria and for the level of sugar, chloride, and protein.

In certain operations in which general anesthesia is contraindicated, local anesthetic fluid can be injected through the spinal tap needle into the epidural or subarachnoid space, producing what is known as a sacral or lumbar block. The anesthetist employs techniques that prevent the anesthetic fluid from flowing up the vertebral canal and silencing nerves to vital organs.

Increased Intracranial Pressure

Many pathologic conditions can produce an increase in intracranial pressure. This is often indicated by a patient's complaining of headaches, but it must be emphasized that most headaches are not the result of an increase in pressure. Increased intracranial pressure may be detected by looking with an ophthalmoscope into the patient's eye and observing the presence of papilledema of the optic disc of the retina. Normally, the optic disc is sharp and distinct, but when there is an increase in CSF pressure, papilledema results. The borders of the disc become blurred, there is congestion or hemorrhage of the peripapillary veins, and the entire optic disc may bulge. The underlying cause should be sought and the pressure relieved in order to prevent brain damage, coma, or death.

After a spinal tap, some of the CSF may leak out into the surrounding tissues. This produces a decrease in intracranial pressure, which is often accompanied by headaches.

Chapter 23

PATHOLOGIC CONDITIONS OF THE CENTRAL NERVOUS SYSTEM

This chapter is designed to give the reader a short introductory overview of the most common neuropathologic conditions that he or she will encounter. In no way is it a substitute for a more detailed reading of the subject.

CEREBROVASCULAR ACCIDENT

Cerebrovascular accident (CVA) may be defined as damage to the brain as a result of a pathologic condition of the blood vessels, especially the arteries. CVAs, or strokes, are the third most common cause of death in the United States, after heart attacks and cancer.

As mentioned in Chapter 1, the brain is very sensitive to oxygen deprivation. If the arterial supply to an area is cut off, it will undergo a process of degeneration and death, producing what is known as an infarcted area (see Appendix III, Figure 9). The arterial blockage may be the result of thrombus formation, emboli, or vasospasm. The clinical picture that one sees depends on the area that is affected, but what is seen most often is some kind of upper motor neuron paralysis.

As one gets older the arteries become less elastic and more fragile; these changes, often combined with hypertension, can result in the rupture of a vessel. The subsequent *hemorrhage* (see Appendix III, Figure 10) results in rapid death or permanent disablement.

An *aneurysm* is a local ballooning out of an arterial wall. Aneurysms in the brain are most often located in and around the circle of Willis, with 50% arising from the internal carotid or middle meningeal arteries. If an aneurysm ruptures, there will be hemorrhaging into the subarachnoid space, followed most often by rapid death.

INFECTIONS

Bacterial Infections

Meningitis is an infection of the cerebral and/or spinal meninges (usually the arachnoid and pia). In 80–90% of cases it is caused by one of three bacteria: *Neisseria meningitidis, Diplococcus pneumoniae,* or *Haemophilus influenzae.* The triad of fever, headache, and stiff neck should immediately alert the physician to the possibility of the disease, and a spinal tap (lumbar puncture) should be done at one.

Brain abscess is a pocket or pockets of pus that have formed in the brain tissue (see Appendix III, Figure 12). The bacteria—most often streptococci—usually have migrated to the brain via the bloodstream from a primary infection elsewhere in the body.

Tetanus occurs when a cut or wound is invaded by anaerobic *Clostridium tetani* or its spores. Once in the tissue, the bacteria produce a powerful neurotoxin which causes severe muscular spasms. The best "cure" is prevention by immunization.

Infection of the dural venous sinuses, like brain abscesses, is most often secondary to an infection elsewhere in the body. Because of its location and the structures in it, infection of the cavernous sinus at the base of the brain is particularly dangerous.

Syphilis is caused by the spirochete *Treponema pallidum.* The last or tertiary stage of the illness

occurs many years after the initial infection and may affect the nervous system, causing a multiplicity of symptoms, many of them mental.

Viral Infections

Encephalitis is inflammation and infection of the brain tissue by any one of a large number of viruses. Among the most common types of encephalitis are St. Louis encephalitis, Eastern encephalitis, Japanese B encephalitis, and encephalitis lethargica. Mortality rates vary from type to type.

Poliomyelitis, once a highly prevalent and dreaded disease, specifically attacks the cell bodies of lower motor neurons, leaving in its wake a trail of death or paralysis. In the developed countries it has been eliminated by a vaccine developed by the great work of Enders, Salk, and Sabin.

Throughout history, *rabies* has been one of the most feared diseases, because once infected the patient always died, with death being preceded by the most terrifying symptoms, including excitability, refusal to drink water (hydrophobia) because of painful laryngeal spasms, and convulsions. The disease is transmitted via the saliva of a warm-blooded animal, such as a dog, cat, squirrel, or fox, that has been infected. The incubation period varies from 10 days to many months, and sometimes more than a year. Animals that have bitten humans should be quarantined for 10 days. If the animal does not die during this period, it is not rabid. If the animal does die, the brain should be examined. Treatment consists of vaccination, but once symptoms have set in there is no cure or hope.

Fungus Infections

Fungal infections of the CNS are rare, but once established they are difficult to treat and the mortality rate is high.

BRAIN TUMORS (NEOPLASMS)

Brain tumors may be derived from: 1) nervous tissue, 2) nonnervous tissue of the CNS, or 3) from primary sites outside the CNS. Tumors of the last-named type are called metastatic in origin (see Appendix III, Figure 15). Malignancy does not depend only on the histology of the tumor cell but also on the location of the tumor. For example, a growth that is benign from a morphologic point of view may be so located that it cannot be reached surgically (e.g., a tumor in the midbrain) and will therefore be fatal.

Because neurons do not undergo mitosis, the vast majority of tumors derived from nervous tissue are glial in origin and are known as *gliomas*. Histologically they may be astrocytomas (see Appendix III, Figure 14), oligodendrogliomas, or ependymomas, which are derived from the ependymal cells. Very rarely some neurons which haven't matured may give rise to *neuroblastomas*. Last are the *medulloblastomas*, which are believed to arise from the granular cells of the external layer of the cerebellum.

Tumors may also arise from nonnervous tissues of the CNS. Those arising from the meninges are known as *meningiomas* (see Appendix III, Figure 13), whereas those from the blood vessels are *hemangiomas*.

Lastly, tumors of the brain may be metastatic. In such a case, the main site of growth is in an organ such as the lung or prostate, from which tumor cells have broken off and reached the brain via the circulatory system.

In the past the prognosis of brain tumors was poor, but with the development of better surgical techniques and earlier detection it has become better.

TRAUMA

In modern medical practice it is common to see trauma to the brain and/or spinal cord from various causes such as accidents (e.g., automobile, work, home, sports) and violence (e.g., blows, bullets, knife wounds). In these cases the skull and/or vertebral column is often fractured, and the delicate nervous tissue is compressed, lacerated, or destroyed, leading to death or permanent neurologic disabilities. Trauma to the brain is often accompanied by tearing of blood vessels, followed by hemorrhage and all the damage that it can produce.

Following an accident, clear or pink-tinged fluid seeping from the ear or nose often indicates a skull fracture with escaping CSF. A skull fracture may exist without escaping CSF, however. Therefore, x-rays must be taken for confirmation.

DEMYELINATING DISEASES

In this group one finds multiple sclerosis (MS), which was discussed in Chapter 1. Also included is *postvaccinal encephalomyelitis*, which may occur, as its name indicates, after an individual has received a vaccination.

DEGENERATIVE DISEASES

Parkinson's disease is the result of a degenerative process that occurs in the basal ganglia and substantia nigra. It affects older people and its cause is unknown. *Alzheimer's disease* is another common condition that affects older people. There is widespread neuronal death, and the cerebral cortex undergoes marked atrophy with a widening of the sulci. This disease produces aphasias or convulsions and a slowly progressive dementia.

TOXINS AND DRUGS

In this category are many exotic substances that are more often found in mystery and espionage stories than in real life, but when found in the latter they often outdo fiction.

Deaths from the venom of snakes, spiders, and scorpions are rare but always stir the imagination. A few years ago in California, a man was convicted of attempted murder for placing a rattlesnake in the mailbox of his intended victim, who barely survived after reaching in to get the morning mail.* Some venoms are neurotoxins which depress the cardiac and/or respiratory centers, whereas others prevent the transmission of nervous impulses.

Heavy metals can also be lethal. Lead poisoning occurs most often in children, especially those in poor urban areas, who often chew on lead-based paint that has peeled from walls. Symptoms occur a few weeks after ingestion and are varied, insidious, and nonspecific. The symptoms may include loss of appetite, irritability, loss of alertness, and, later, drowsiness, seizures, and coma. Mercury, manganese, and arsenic as well as many industrial compounds can cause neuropathy. For this reason, an accurate, detailed history of a patient's occupation, habitat, and other factors is very important. Finally, there are plant neurotoxins from curare, ergot, and some species of mushrooms.

Fifteen thousand people in the United States die every year from barbiturate poisoning—it is the leading cause of toxic death. By contrast, in 1979 there were only five reported cases of rabies, yet rabies is a far more terrifying word than barbiturates. Because barbiturate deaths are mostly self-inflicted, it would be far better if physicians would instead prescribe chloral hydrate, because it is one of the best and safest hypnotics.

So much has already been written about heroin and cocaine that nothing can be added here that will enlighten or help the reader.

ALCOHOLISM

Alcoholism is one of the most prevalent diseases in the United States: an estimated 12–15 million people are alcoholics or have a drinking problem. Alcoholism has many causes and a strong genetic predisposition is involved.** Most of us have seen the havoc it wreaks upon the individual, the family, and society. There is no known cure, but complete abstinence and joining Alcoholics Anonymous give the best chance for avoidance of relapse.

EPILEPSY

This is a common neurologic condition that affects over 1 million people in the United States. Once—and perhaps even now—a sense of shame was attached to epilepsy, but it is a disease like any other.

Epileptic seizures have many forms. The grand mal type is characterized by a sudden, massive, uncoordinated electrical discharge from cerebral neurons, resulting in loss of consciousness and convulsive movement of body musculature. Petit mal epilepsy occurs most often in children. The seizures consist of a loss of consciousness lasting from 1–3 seconds without closure of the eyes or any muscle spasms. In fact, the child is often unaware of his or her condition, and the child's teachers may complain that the pupil often doesn't pay attention.

Although much research has been and is being done, the cause of this disease is still unknown. Fortunately, in the majority of cases epileptic seizures can be prevented completely by the use of proper anticonvulsive drugs.

METABOLIC DISEASES

Metabolic diseases of the CNS are divided into two main groups: the acquired and the inherited. The former are really metabolic diseases of other organs that secondarily affect the brain. For example, hypothyroidism in children (cretinism) pro-

*The author disclaims responsibility for planting this idea, or any like it, in the minds of his readers.

**An interesting research paper on this topic is: Goodwin, D., et al. Alcohol problems in adoptees raised apart from alcoholic parents. Archives of General Psychiatry 28:238, 1973.

duces, among other things, severe mental retardation. Hypoglycemia can also adversely affect the brain. Recently in New York City a man was convicted of having attempted to murder his wealthy socialite wife by injecting her with a massive dose of insulin. She survived, but is in a coma and doctors fear she will remain so for the rest of her life. Because the liver is a vital organ involved in the metabolic process, damage to it can produce CNS symptoms that may end in coma and death.

The inherited or inborn metabolic diseases are due to an enzyme defect that produces an abnormal metabolism of various substances. As there are now over 100 such diseases, many with quaint names such as gargoylism and maple syrup urine disease, only the most common are mentioned here. In *phenylketonuria* there is an absence of the enzyme phenylalanine hydralase, which normally converts phenylalanine to tyrosine. In its absence, and if not detected and treated in time, the phenylalanine accumulates in the body and will produce neurologic and mental symptoms.

Tay-Sachs disease is seen almost exclusively (95%) in Jewish children with an Eastern European background. Due to an absence of hexosaminidase A, there is an accumulation of lipids known as gangliosides in the brain cells. The disease begins in infancy and, there being no known means of prevention or cure, ends fatally by age 3 or 4.

Infantile Goucher's disease manifests itself in the first half-year of life. Due to faulty fat metabolism, there is a buildup of cerebrosides in the cells of bone marrow, liver, and spleen, as well as in the brain. There is no known cure and the disease runs a progressively downhill course.

CONGENITAL DEFECTS

Congenital neuropathologic conditions may arise from a number of different causes, including genetic disorder, radiation, anoxia, and maternal infections. *Down's syndrome (mongoloidism)* is due to genetic disorder in the child, and there is a much higher incidence of it in women over 40 who become pregnant.

A pregnant woman may get a rubella infection, which is a very mild thing for her. However, the virus may also attack the fetus, causing the child to be born deaf. In a pregnant woman who is also a drug addict, the heroin passes the placental barrier and the unborn child may become addicted. After birth, the child is cut off from the source of the drug, and severe withdrawal signs are seen.

Finally, there are many neurologically relevant conditions, such as mental retardation, failure of the brain to develop (anencephaly), and hyperactivity, for which the exact cause is unknown.

Appendix I

SPECIAL NEUROANATOMICAL AND NEUROPHARMACOLOGIC GLOSSARY

WORD	DERIVATION	ILLUSTRATIVE EXAMPLE OR COGNATE
Agnosia	*a,* not *gnosis,* knowledge	Agnostic
Alexia	*a,* not *lexis,* word	Lexicon
Aqueduct	*aqua,* water *ductus,* a leading	Aquarium; duke, "a leader"
Arachnoid	*arachne,* spider *eidos,* resemblance	The arachnoid resembles a cobweb.
Arcuate	*arcus,* a bow	Arch; archery
Astrocyte	*astron,* star *kytos,* cell	Astronomy
Brachium	*brachium,* arm	Embrace
Carotid	*karoo,* to put to sleep	Pressure on the carotid artery results in unconsciousness, as is well known in judo.
Caudate	*cauda,* a tail	A caudate nucleus has a tail.
Cerebellum	*Cerebellum* is the diminutive of *cerebrum* (brain), and it means "little brain."	
Cerebrum	*cerebrum,* brain	Cerebration is thinking.
Chiasma	The Greek letter chi (χ) is cross-shaped.	A chiasma is an arrangement in the form of a crossing.
Chorea	*choreia,* dance	People afflicted with Huntington's chorea exhibit characteristic ("dancing") movements. Choreography
Cistern	*cisterna,* a well	
Claustrum	*claustrum,* enclosure	Claustrophobia; closet
Coronary	*corona,* crown or garland Also, the *corona radiata* is a fan-shaped ("radiating") fiber mass in the cerebral cortex.	The coronary arteries encircle the heart. Coronation
Corpus callosum	*corpus,* body *callosum,* hard	Callus; corporation
Cortex	*cortex,* bark	The cerebral cortex covers the cerebral hemisphere, much as bark covers the trunk of a tree.
Cuneate	*cuneatus,* wedge-shaped	The cuneiform writing of ancient Babylon had wedge-shaped characters.
Decussation	The Roman numeral X is called *deca.*	A decussation is a crossing.
Dendrite	*dendron,* branching figure or tree	Rhododendron
Dentate	*dens,* tooth *dentatus,* tooth-shaped	Dentist
Dura mater	*dura,* hard *mater,* mother	Durable; alma mater
Epi-	Greek prefix meaning "upon, over, above"	An epitaph is inscribed over a grave *(taphos).*

WORD	DERIVATION	ILLUSTRATIVE EXAMPLE OR COGNATE
Fasciculus	*fasciculus,* a bundle (of rods or fibers)	The symbol of the Italian Fascists was the Roman bundles of rods, the *fascis* seen on old Mercury head dimes.
Fornix	*fornix,* arch	In ancient Rome the prostitutes hung around the supporting arches of the viaducts. A man visiting the area was engaged in fornication.
Genu	*genu,* knee	Genuflect, i.e., bend (bow) before royalty
Glia	*glia,* glue	Glial cells "hold together" the neurons.
Glossal	*glossa,* tongue	Glossary
Gracilis	*gracilis,* slender	
Gyrus	*gyros,* ring, circle	Gyrate; gyroscope
Hippocampus	*hippus,* horse *campus,* sea	In cross-section the hippocampus resembles a seahorse. Hippodrome
Hypo-	Greek prefix meaning "under, below"	A hypodermic goes under the skin *(dermis).*
Insula	*insula,* island	Insulin is produced by the islands of Langerhans; insulation
Internuncial	*inter,* between *nuncio,* messenger	Announce; papal nuncio
Lemniscus	*lemniscus,* ribbon, band	
Lentiform	*lentiformis,* lens-shaped	*Lens* is the Latin word for lentil, the "lens-shaped" vegetable of the bean family.
Limbic	*limbus,* border or edge	Limbo is the area bordering on Hell.
Lingula	*lingula,* little tongue	Linguist; language
Lumbar	*lumbus,* loin or flank	Lumbago
Mamillary	*mamma,* breast	Mammary; mammals
Mesencephalon	*meso,* middle *encephalos,* the brain	Mezzanine
Oligodendroglia	*oligo,* few *dendron,* branching figure or tree *glia,* glue	Oligarchy, "a few who rule"
Pallidus	*pallidus,* pale	The pallidus is pale in comparison to the neighboring putamen.
Peduncle	*ped,* foot, limb, stalk	Pedal; pedestrian
Pia mater	*pia,* soft, delicate *mater,* mother	Pianissimo is a musical term meaning "very soft."
Pineal	*pinea,* pine cone	The pineal body is conical.
Pons	*pons,* bridge	Pontoon
Ramus	*ramus,* branch	Ramifications
Rectus	*rectus,* straight	Rectify; erect
Reticular	*reticulum,* small net	A reticle is the network of lines in a telescopic sight; a lady's reticule is a small net bag.
Rhinencephalon	*rhin,* nose *encephalos,* the brain	Rhinoceros
Rubro	*ruber,* red	Ruby
Sacral	*sacer,* holy, sacred	The sacral bone was believed to resist decomposition and thus serve as the basis for resurrection.
Sagittal	*sagitta,* arrow	Sagittarius is the Archer of the zodiac.
Sella turcica	*sella,* saddle *turcica,* Turkish	The sella turcica resembles a Turkish saddle.
Septum	*septum,* a partition	Separate
Substantia nigra	*substantia,* substance *nigra,* black	Nigeria; negroid
Tapetum	*tapete,* carpet	Tapestry
Tectum	*tectum,* roof	Architecture
Temporal	*tempus,* time	The temporal area gives evidence of the passage of time; i.e., the hair turns gray.
Tentorium	*tentorium,* tent	
Tubercle	*tuber,* a swelling or rounded projection	Tubers (potatoes); protuberance
Vagus	*vagus,* wandering	The vagus nerve extends into the thorax and abdomen. Vagabond; vagrant

WORD	DERIVATION	ILLUSTRATIVE EXAMPLE OR COGNATE
Velum	*velum,* covering	Veil
Ventricle	*ventrus,* chamber, cavity, hollow, stomach	A ventriloquist "speaks from the stomach."
Vermis	*vermis,* worm	The cerebellar vermis resembles a worm. Vermin

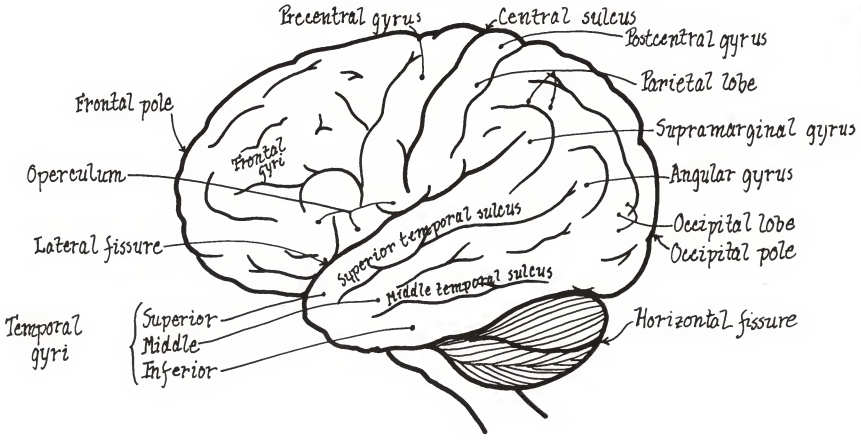
TERMS RELATED TO NEUROPHARMACOLOGY

Barbiturate	Emil Fisher first produced barbiturates by condensing malonic acid with urea. The correct name should be malonylurate. The urea was extracted from large quantities of urine given to him by a waitress who worked in a coffeehouse he frequented (coffee is a diuretic agent). Not surprisingly, her name was Barbara, and to thank her for her efforts on behalf of science, he named the new drug after her.
Belladonna	*Bella donna* is Italian for "beautiful lady." In Italy during the Renaissance, ladies before going to parties would put belladonna (atropine) in their eyes, causing the pupils to dilate and the eyes to sparkle, thus enhancing their beauty. However, the drug greatly blurred their vision, and one can imagine the scene that ensued when such a lady mistook her husband for her lover.
Cocaine	Cocaine comes from the leaves of the coca tree. Sigmund Freud discovered its use as a local anesthetic for the eye. For a while, he also used it to get "high," or "euphoric," as he put it.
Hashish	This is an Arabic word. In the Middle East during the Crusades, professional killers often smoked hashish before doing a "hit," and for this reason they were called *hash-ha-shans.* The Crusaders could not pronounce this guttural word and corrupted it to "assassins," from which we get the word "assassinate."
Heroin	This drug gets its name from the fact that it often gives one transitory heroic feelings.
Marijuana	This drug is so called from the belief that it is an aphrodisiac. It is derived from the Spanish names Maria, which is feminine, and Juan, which is masculine.
Morphine	Morpheus was the Greek god of dreams, and taking morphine puts one in a dreamlike state. Shapes and forms appear in dreams, and from this is also derived the word morphology, meaning the study of structures or forms.
Nicotine	This drug was named after Jean Nicot, who introduced tobacco into France.

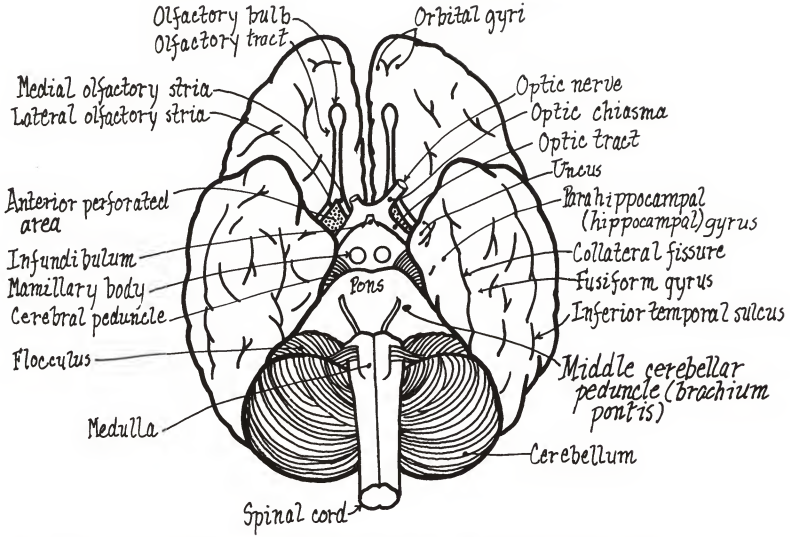
Appendix II

ATLAS OF THE BRAIN

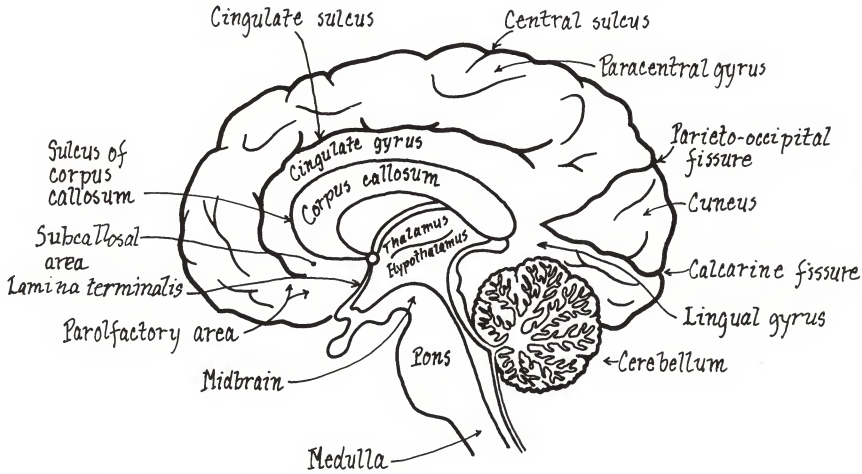
Atlas Plate I Lateral view of the brain.



Atlas Plate II Basal view of the brain.*

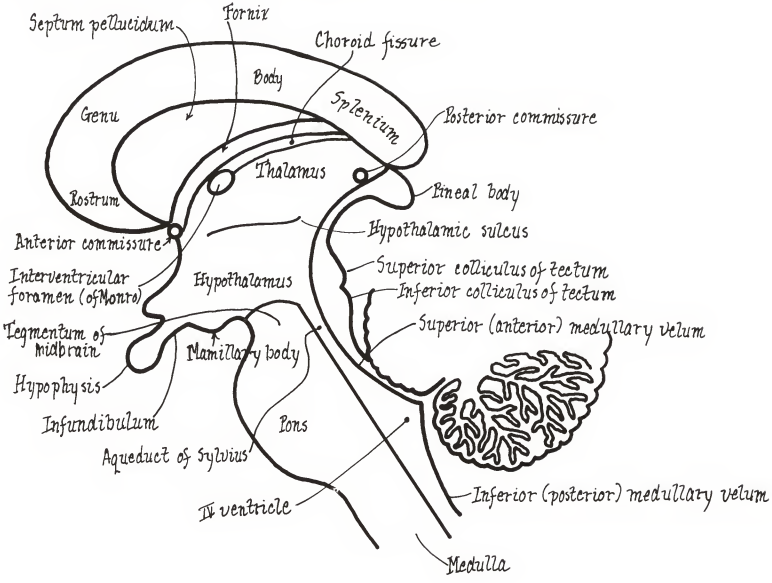


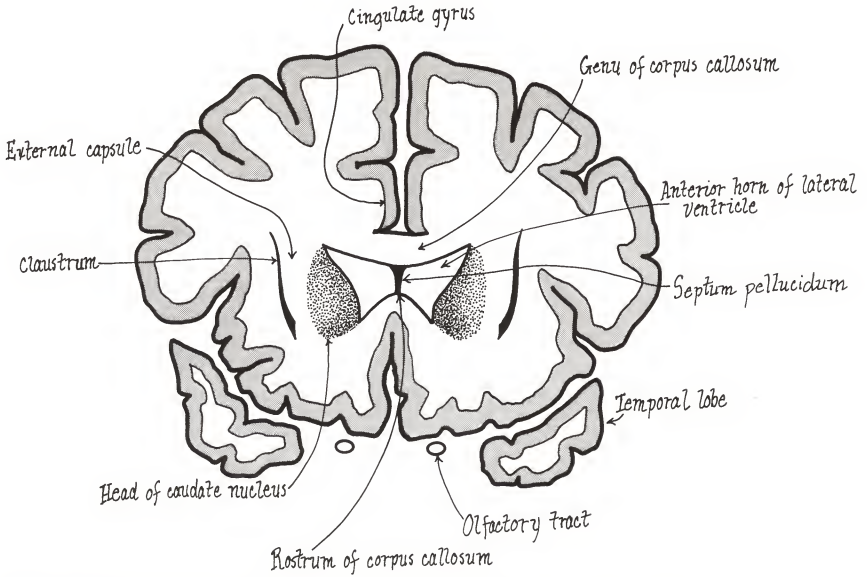
*For a more detailed view of the brainstem with the cranial nerves, see Plate VIII of this atlas.



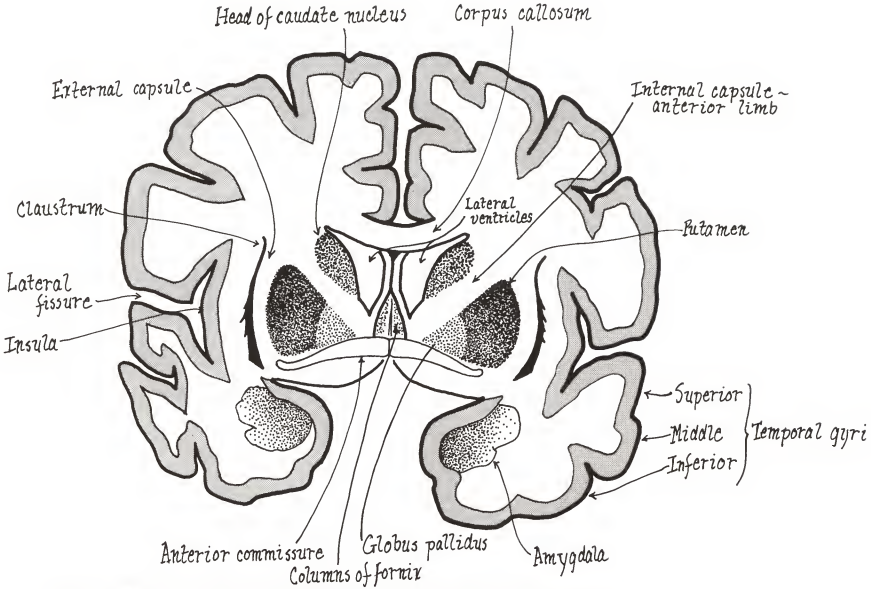
ATLAS PLATE III. MID-SAGITTAL VIEW OF THE BRAIN.

ATLAS PLATE IV. ENLARGED MID-SAGITTAL VIEW OF THE BRAIN.

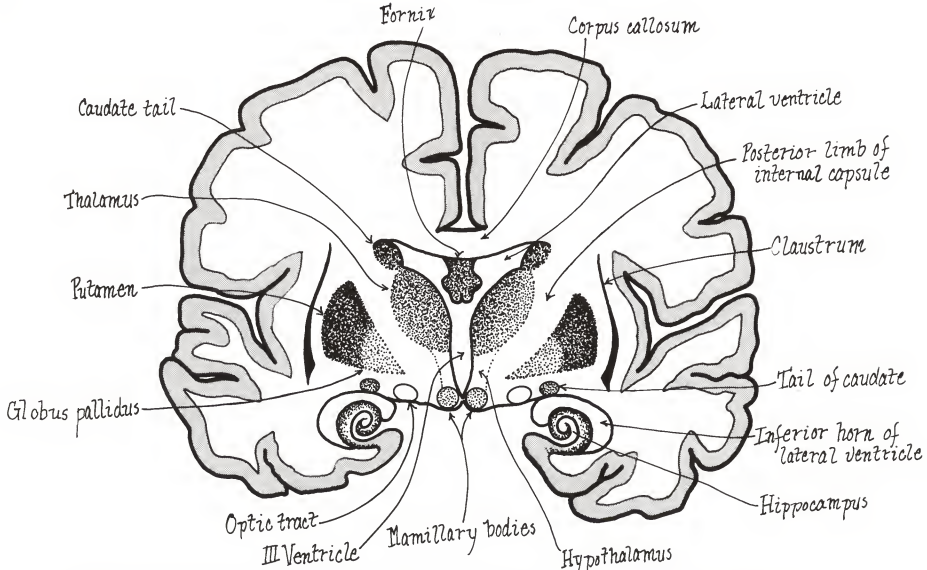




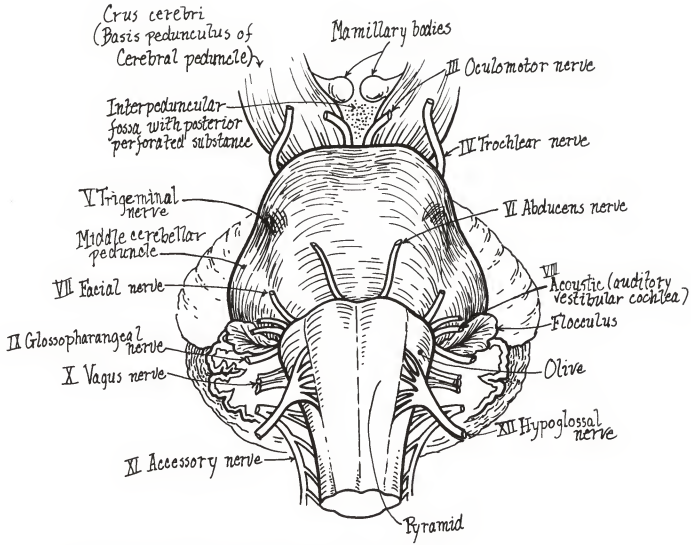
ATLAS PLATE V. CROSS-SECTION OF THE BRAIN, ANTERIOR TO THE ANTERIOR COMMISSURE.



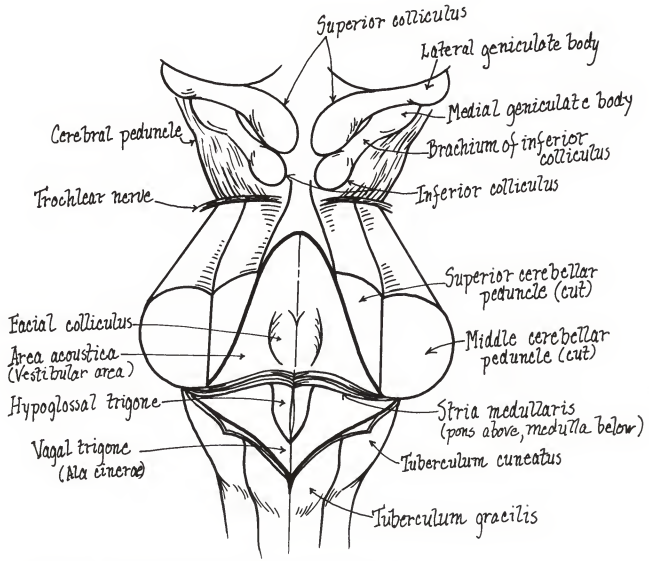
ATLAS PLATE VI CROSS-SECTION AT THE LEVEL OF ANTERIOR COMMISSURE.



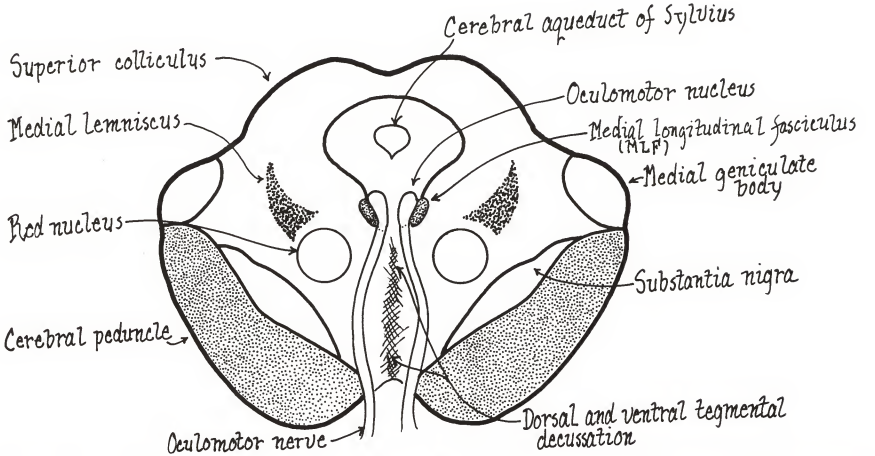
ATLAS PLATE VII. CROSS-SECTION OF THE BRAIN AT THE LEVEL OF THE MAMILLARY BODIES.



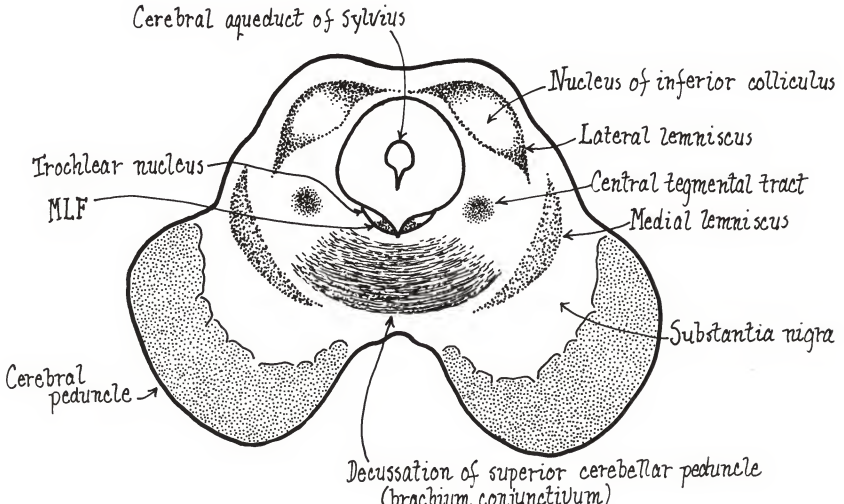
ATLAS PLATE VIII. VENTRAL VIEW OF THE BRAINSTEM.



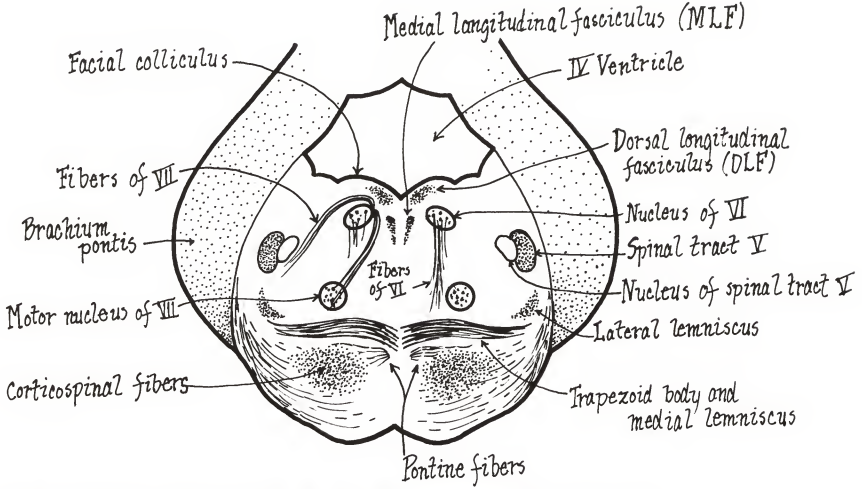
ATLAS PLATE IX. DORSAL VIEW OF THE BRAINSTEM.



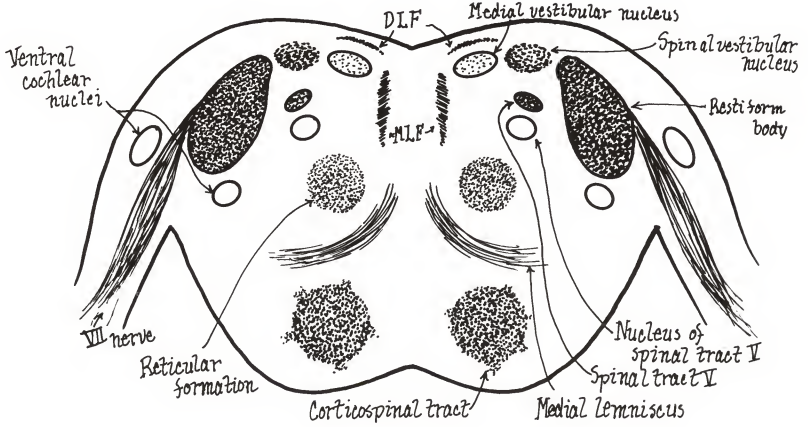
ATLAS PLATE X CROSS-SECTION OF THE MIDBRAIN AT THE LEVEL OF THE SUPERIOR COLLICULUS.



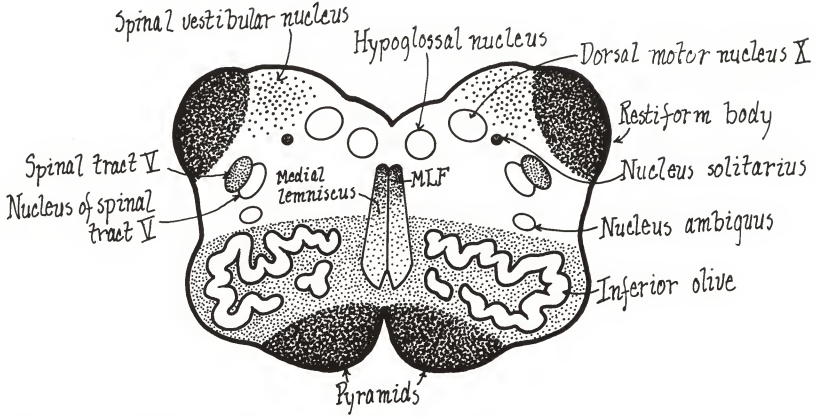
ATLAS PLATE XI. CROSS-SECTION OF THE MIDBRAIN AT THE LEVEL OF THE INFERIOR COLLICULUS.



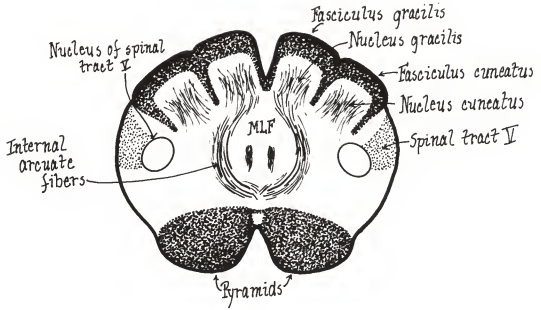
ATLAS PLATE XII. CROSS-SECTION OF THE PONS AT THE FACIAL COLLICULUS.



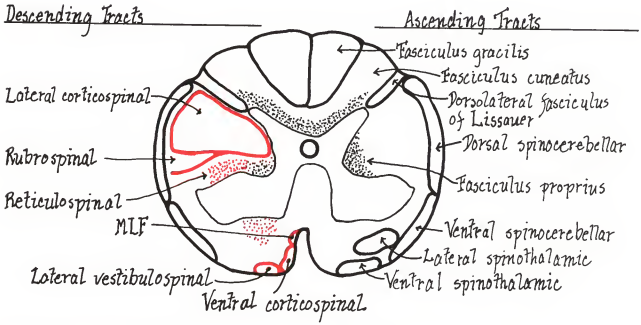
ATLAS PLATE XIII. CROSS-SECTION OF LOWER PONS.



ATLAS PLATE XIV. SECTION THROUGH UPPER MEDULLA.



ATLAS PLATE XV. SECTION THROUGH LOWER MEDULLA.



ATLAS PLATE XVI. SECTION THROUGH SPINAL CORD AT MIDCERVICAL LEVEL.

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Appendix III

ATLAS OF NORMAL AND PATHOLOGIC COMPUTERIZED TOMOGRAPHY SCANS OF THE BRAIN

Prepared by Rina Tadmor, M.D.

The discovery of x-rays by William Roentgen in 1895 was one of the great contributions to medicine. A further revolutionary step was made in 1972 by the physicist Godfrey Hounsfield and the neuroradiologist James Ambrose, who introduced the technique of computerized tomography (CT). They, like Roentgen, won the Nobel Prize in Medicine for their work.

In conventional radiology, most of the shadows and outlines of the various three-dimensional structures are superimposed on a two-dimensional film. Further, in conventional x-rays of the skull, the brain is not seen because of its low density. However, CT is about 100 times more sensitive than conventional radiography, and enables one to see clearly the brain and its subdivisions. CT images appear in the various shades of gray from black to white. In the negative mode (the one most commonly used in CT) the densest objects and materials, such as bone, appear as white, whereas those of low density such as cerebrospinal fluid are black. The ventricular system, the cerebral sulci, and similar structures are thus rendered quite visible. This mode of presenting the CT image may at first present some confusion, as the gray matter in the basal ganglia, cerebral cortex, and elsewhere appears white on a CT scan, because it is packed with cell bodies and is therefore relatively dense. The white matter is lower in density and therefore shows up as gray or black.

A complete CT examination of the brain involves 10–12 successive, parallel, horizontal scans or "cuts" of the brain. The injection of intravenous contrast substances enhances the visibility of lesions and thereby facilitates the viewing of pathologic conditions such as tumors and abscesses.

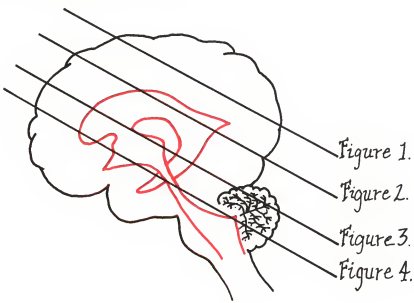
This atlas attempts to give a basic understanding of normal and abnormal CT scans. With this foundation, the student can go on to further reading and practice in interpreting more detailed scans. Figures 1–4 in this atlas are normal horizontal scans. The drawing on this page indicates the level at which each cut was taken. The ventricular system is outlined in red in the drawing; it appears in black in the CT images themselves. Figure 5 is a coronal scan, and Figures 6 and 7 are partial horizontal scans showing the orbital areas and the structures of the ear, respectively. The eight pathologic scans in Figures 8–15 represent some of the most common disease conditions seen in the neurologic service.

Finally, listed below are three important works related to CT which will give you a deeper understanding of this very important subject:

Kieffer, S. A., and Heitzman, E. R., *Atlas of Cross-Sectional Computed Tomography, Ultrasound, Radiography, Gross Anatomy*. Harper & Row, New York, 1979.

Harwood-Nash, D. C., *Neuroradiology in Infants and Children*, Volume II, pp. 461–504. C. V. Mosby, St. Louis, 1976.

Gonzales, C. F., Grossman, C., and Palacios, J., *Computed Brain and Orbital Tomography: Technique and Interpretation*. John Wiley & Sons, New York, 1976.



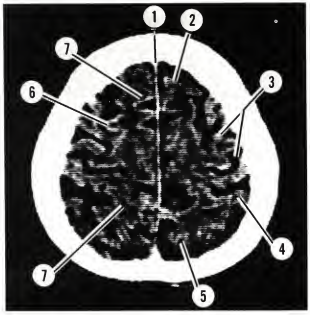


Figure 1. Horizontal section of the superior part of the normal brain.

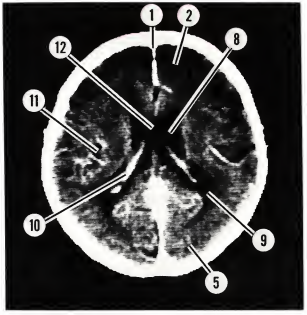


Figure 2. Horizontal (axial) section at a lower (inferior) level.

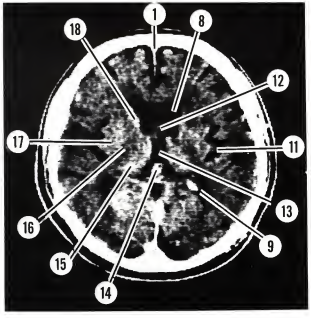


Figure 3. Horizontal section at a still lower level.

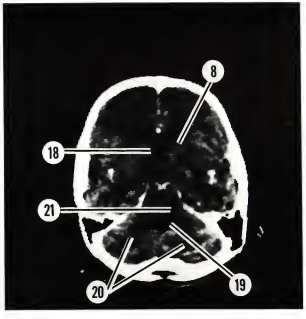


Figure 4. Horizontal section through the basal part of the brain.

Legend: *1*, falx cerebrum; *2*, frontal lobe; *3*, sulcus; *4*, gyrus; *5*, occipital lobe; *6*, gray matter; *7*, white matter; *8*, anterior horn of lateral ventricle; *9*, posterior horn of lateral ventricle; *10*, choroid plexus; *11*, insula; *12*, septum pellucidum; *13*, third ventricle; *14*, calcified pineal body; *15*, thalamus; *16*, internal capsule; *17*, lentiform nucleus; *18*, head of caudate nucleus; *19*, fourth ventricle; *20*, cerebellar hemisphere; *21*, pons.

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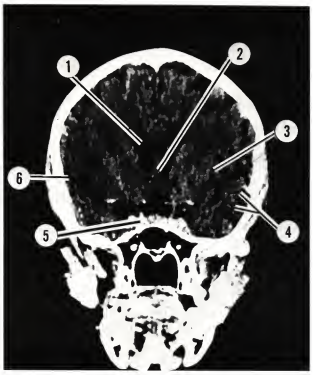

Figure 5. Coronal section of the brain 1 inch anterior to the external auditory meatus. *1*, lateral ventricle; *2*, septum pellucidum; *3*, insula; *4*, temporal lobe; *5*, cavernous sinus; *6*, lateral fissure.

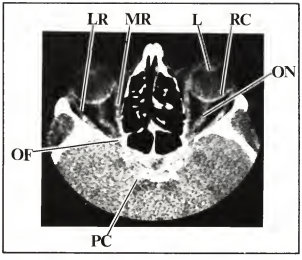

Figure 6. Horizontal cut through the middle of the orbits. *L*, lens; *RC*, retina and choroid; *ON*, optic nerve; *LR*, lateralis rectus muscle; *MR*, medial rectus muscle; *OF*, optic foramen; *PC*, posterior clinoid process of the sella turcica.

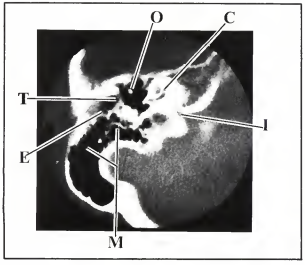

Figure 7. Horizontal section through the middle and inner ear. *E*, external auditory canal; *T*, tympanic membrane (eardrum); *O*, ossicle in middle ear cavity; *C*, cochlea in inner ear cavity; *I*, internal auditory canal; *M*, mastoid air cells.

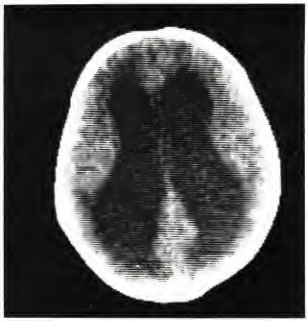

Figure 8. Hydrocephalus. The accumulated cerebrospinal fluid has greatly increased the size of the lateral ventricles.

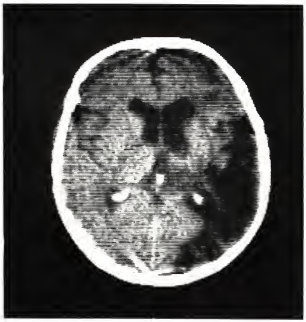

Figure 9. Infarct. Obstruction of the right middle cerebral artery has produced a dark infarcted area in the parieto-occipital lobes with no displacement of brain structures.

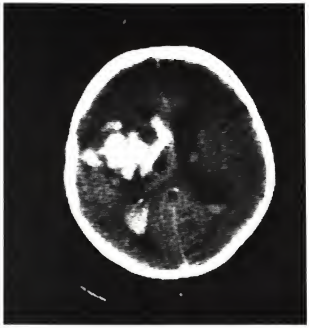

Figure 10. Intracerebral hemorrhage. The dense, white, irregular mass in the left parietal lobe represents blood, which has also ruptured into the lateral ventricle.

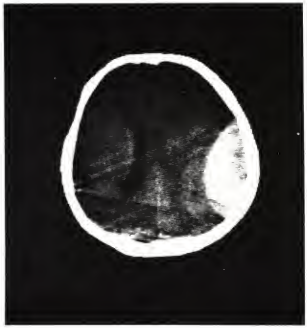

Figure 11. Epidural hematoma. The well delineated white area represents an extracerebral hemorrhage in the right parietal region, causing compression and closure of the lateral ventricle on the same side.

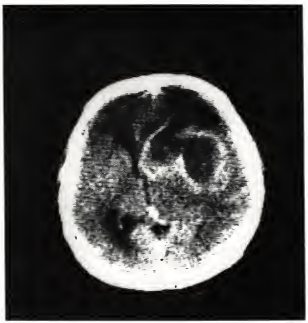

Figure 12. Abscess. A multilobulated mass in the frontoparietal lobes has caused massive displacement of brain tissues and structure.

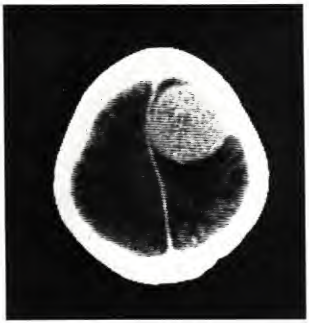

Figure 13. Meningioma. The round, white, homogeneous tumor in the right frontal lobe has caused a displacement of the falx cerebrum.

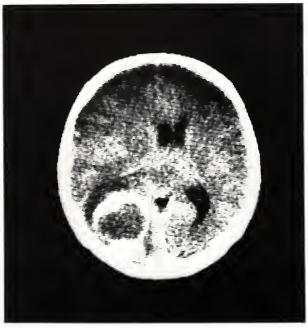

Figure 14. Astrocytoma. This round, well delineated, nonhomogeneous tumor in the left parieto-occipital lobes is surrounded by a dark edematous area. This growth has caused a shift in structures as seen by the displaced septum pellucidum and obliteration of the posterior horn of the lateral ventricle.

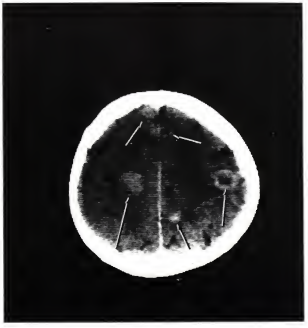

Figure 15. Metastasis. Multiple round lesions (arrows) in both hemispheres represent metastatic spread ("seeding") from a primary tumor elsewhere in the body.

Appendix IV

SAMPLE EXAMINATION QUESTIONS

1. Which of the following tracts or pathways is completely uncrossed in its entire course?
   a. Pain and temperature from the face
   b. Proprioception from the body
   c. Corticospinal
   d. Dorsal spinocerebellar
   e. Vestibular pathways
2. What condition results if the right optic nerve is cut?
   a. Left homonymous hemianopsia
   b. Bitemporal hemianopsia
   c. Right homonymous hemianopsia
   d. Binasal hemianopsia
   e. None of the above
3. Which of the following is not true concerning the hypothalamus?
   a. It is concerned with temperature regulation
   b. It has a hunger center
   c. It is concerned with equilibrium
   d. It influences pituitary secretions
   e. It has areas concerned with emotional reactions
4. Which of the following statements concerning the neuron is not true?
   a. It is very sensitive to oxygen deprivation
   b. If the axon is cut the cell body will always die
   c. Myelin is laid down by the sheath of Schwann in the PNS
   d. Nissl bodies are found in the cytoplasm of the cell body
   e. Mature neurons don't undergo mitosis
5. Which of the following tracts doesn't synapse on the final common pathway?
   a. Rubrospinal tract
   b. Corticospinal tract
   c. Spinothalamic tract
   d. Vestibulospinal tract
   e. All of the above
6. Which of the following is not a sign of cerebellar injury?
   a. Uncoordinated movements
   b. Dizziness
   c. Athetosis
   d. Falling
   e. Intention tremor

In the following three questions match the tract with the peduncle in which it runs:

7. Corticopontocerebellar tract
8. Dentorubrothalamic tract
9. Vestibulocerebellar tract
   a. Superior cerebellar peduncle
   b. Middle cerebellar peduncle
   c. Inferior cerebellar peduncle
10. Obstruction of the left anterior cerebral artery past the anterior communicating artery is likely to cause defective movement or paralysis in the:
    a. Right lower limb
    b. Right upper limb
    c. Muscles of the face on the left side
    d. Muscles of the face on the right side
    e. Left lower limb
11. With respect to the subcortical motor areas (i.e., basal ganglia, etc.) all of the following statements are true except:
    a. Damage to them can result in pill-rolling tremor
    b. They aren't connected to the cerebral cortex
    c. They are part of the extrapyramidal system
    d. They are connected with the red nucleus
    e. They are connected with the thalamus
12. A patient exhibits bitemporal hemianopsia. In which of the following areas is the lesion most likely to be?
    a. Lateral geniculate body
    b. Midline of the optic chiasma
    c. Optic radiations
    d. Visual cortex
    e. Optic tract
13. Which of the following don't synapse in the thalamus?
    a. Pain and temperature from the face
    b. Fibers from the dentate nucleus
    c. Proprioception fibers from the body
    d. Auditory fibers
    e. Pressure and touch fibers from the face
14. A patient suffers from an upper neuron paralysis that affects his arm. The lesion can be in any of the following areas except:
    a. Motor cortex
    b. Internal capsule
    c. Crus cerebri (basis pedunculi)
    d. Tegmentum of the midbrain
    e. Pyramid
    f. Lateral white column of the spinal cord
15. If the dorsal spinal root is cut in the sacral region, which of the following would show Wallerian degeneration in the cervical area of the cord?
    a. Spinothalamic tract
    b. Fasciculus cuneatus
    c. Ventral spinocerebellar tract
    d. Lateral corticospinal tract
    e. Fasciculus gracilis
16. The cell bodies of the preganglionic parasympathetic fibers that innervate the descending colon are situated in the:
    a. Dorsal motor nucleus of the vagus
    b. Nucleus ambiguus
    c. Lateral gray column of the spinal cord in the thoracic segments T_{10}–T_{12}

d. Inferior mesenteric ganglion
e. Lateral gray column of the spinal cord in the S_2–S_4 sacral segments

17. A patient has total deafness in the left ear. In which of the following areas is the lesion most likely to be?
    a. Left superior temporal gyrus
    b. Right and left cochlear nuclei
    c. Left auditory nerve
    d. Left lateral lemniscus
    e. Right and left inferior colliculi
18. Examination reveals that a patient doesn't sweat in the area supplied by thoracic spinal nerves T_1–T_2. A lesion in which area *won't* give rise to this condition?
    a. Sympathetic trunk
    b. Intermediate lateral gray column of the spinal cord
    c. Ventral roots of the spinal cord
    d. White rami communicantes
    e. Dorsal roots of spinal nerve
    f. Gray rami communicantes

In the following three questions three common reflexes are listed. Match them with the correct cranial nerves that are involved in the reflex arc.

19. Corneal reflex
20. Horizontal nystagmus
21. Gag reflex
    a. optic-facial
    b. vestibular-facial
    c. glossopharyngeal-vagus
    d. vestibular-oculomotor and abducens
    e. trigeminal-facial
    f. trigeminal-vagus

Answer the following questions by indicating the letter of the statement that correctly applies.

    a. If both statements are true and there is a causal relationship between the two
    b. If both statements are true but there is not causal relationship between the two
    c. If the first statement is true but the second is false
    d. If the first statement is false and the second is true
    e. If both statements are false

22. Most of the spinocerebellar fibers enter the cerebellum on the ipsilateral side, and therefore injury to the right inferior cerebellar peduncle results in a person falling to the right side.
23. Damage to the genu of the left internal capsule results in a paralysis of the entire right side of the face, because the corticobulbar fibers are located in the genu of the internal capsule.
24. If the right thalamus is damaged then the patient will lose all somatic sensations on the left side of the body and face, because these fibers all eventually cross over to the opposite side from which they entered the cord and brainstem.

Answers

1. *d*	6. *c*	11. *b*	16. *e*	21. *c*
2. *e*	7. *b*	12. *b*	17. *c*	22. *a*
3. *c*	8. *a*	13. *d*	18. *e*	23. *d*
4. *b*	9. *c*	14. *d*	19. *e*	24. *e*
5. *c*	10. *a*	15. *e*	20. *d*	

Index

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